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SECTION OF SURGERY

PRESIDING: Dr. Richard H. Miller, Boston, Chairman; Dr. Allen G. Rice, Springfield, Secretary.

CHAIRMAN MILLER: *Gentlemen*—In accordance with time-honored custom, we are gathered here as one group of the Massachusetts Medical Society to discuss certain matters of interest and importance in the art and science of surgery. As a Section we have no particular business to transact aside from the scientific exercises except to elect a Chairman and Secretary to arrange the meeting for next year. It is the custom for the Chairman of the Section to appoint a Nominating Committee of three, who shall later report the names of the men whom they suggest. I will do that later in the morning when more are present.

Your Secretary and I, in arranging this meeting, gave to the matter a great deal of thought and long consideration, and we decided that it was appropriate and feasible that we should go back to the old, trite subject of the appendix. The subjects which have been more discussed of recent years have been drummed into your ears on all sides; you have been "bethumped" by facts about all sorts of surgical matters which have been studied; but all the time patients in our hospitals are dying of appendicitis, and they are dying in numbers greater than before. You know as well as I that if we walk through the wards of any hospital of size in this country we will find one, two or three patients dying of acute appendicitis, and these conditions really should not exist because we know that if we operate in the interval the mortality is extremely low.

The medical profession has been more and more interested in this mortality in appendicitis, and during the past year or two I have observed numerous articles in the daily newspapers on the same subject, showing that the laity also is concerned about this. So your Secretary and I feel that, although we have hit on a very common, everyday matter for discussion, it is a subject of such importance that we can all derive benefit from hearing it discussed today.

The speakers who are going to address you are men of experience, wisdom and judgment from different parts of this state; and we are fortunate also in having a learned man from Philadelphia, Dr. Muller, who will terminate the discussion in the latter part of the morning.

I am going to ask that the different speakers come on to the platform and that all the discussers at least come down in front, also that each discusser will please announce his name and residence when he rises.

The first paper of this meeting will be presented by Dr. Royal P. Watkins, on "Chronic Appendicitis."

CHRONIC APPENDICITIS*

BY ROYAL P. WATKINS, M.D.†

IN preparing this paper one of the first things I discovered was that there have already been about 662 papers published on the same subject. What I have to say, therefore, will probably have been said many times in many different ways, and will doubtless sound very familiar to you all. This will not be because I have read all of these papers, nor because you have read all of them, but because the same problems which present themselves to me present themselves also to you. It would seem that the subject had been discussed enough; that enough had been written about it. The fact that it is still being discussed is evidence that there are at least some things about it that have not been settled to the satisfaction of all and that we still have hope that they may be.

The discussion of the question as to whether there is such a thing as chronic appendicitis is a splitting of hairs. What one pathologist calls

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†Watkins — Surgeon-in-Chief, Fairlawn Hospital, Worcester. For record and address of author see "This Week's Issue," page 379.

a healed appendix, another calls a chronic appendix. What one clinician calls recurrent appendicitis, another calls chronic appendicitis. Both pathologists admit an abnormal process of long duration and both clinicians recognize symptoms of long duration due to a variation from normal in the appendix. For myself I prefer to call it chronic appendicitis. It is sufficiently descriptive and accurate and saves a lot of explanation to the patient of which he has understood nothing when you get through.

There was a time, not so long ago, when every patient with a long-continued or periodically recurring discomfort in the right abdomen; or with troublesome gas in the bowels, especially with constipation; or with indefinite abdominal symptoms called chronic indigestion; in fact every patient with abdominal symptoms which could not be readily accounted for by something else, must have his appendix out on a diagnosis of chronic appendicitis. Most of these appendices the pathologists were kind enough to report as chronic appendicitis, but in a good many of the cases the appendix looked quite normal and the patients were not relieved of their symptoms. This was very disturbing both to the patient and to the surgeon. After a few of these experiences we became more and more wary of making that diagnosis and have now, I believe, gone to the other extreme; we are not willing to operate unless we can satisfy ourselves definitely that the trouble is in the appendix. This may be a proper attitude. It certainly would be if our diagnostic methods had reached the point where we could accurately diagnose all of our abdominal cases. We are making some progress but are a long way from that point yet. Until we reach it, we will continue to have at least an occasional chronic case in which an exploratory operation is justifiable, and however careful and honest we may be, we will occasionally remove a normal appendix.

A chronic appendix demanding operation is one in which any chronic process is going on which interferes with the normal emptying of the organ. The effort of nature is to keep things from going in and to expel them if they do. There is a valve guarding the entrance, ciliated epithelium lining the lumen and waving toward the entrance, and the action of the muscularis is toward the cecum. A narrowing of the lumen at any point by kink, contracture or swelling interferes with the action of these agencies. Whatever is in the canal beyond the point of narrowing is held there. In a longer or shorter time it may cause a pressure necrosis in the mucous membrane. This gives an excellent culture medium for the bacteria and if they take advantage of it a chronic becomes an acute condition. It would seem more reasonable then to say that there is no acute appendicitis without a preceding chronic one, than that there is no such thing as chronic appendicitis. At any rate it is fair to presume both from the course of events enumerated and from the clinical history that *many* of the acute appendices are preceded by a chronic process. Deaver found a fecolith in 22.3 per cent of 120 acute cases. There must of course have been a preceding chronic process in these cases. I have seen no analysis of the histories of acute cases made to bring out this point, but from my own experience I am sure it is very common. In looking over histories and operative findings in our own hospital records, it is apparent that not enough attention has been given to this feature so that it is possible to compile any accurate data about it.

An interesting finding has been given me by Dr. Raymond H. Goodale pathologist at the Worcester City Hospital. I quote from his letter: "Of 975 appendices over a period of two years, I find that we had 59 appendices containing the oxyuris vermicularis. This is an incidence of 6 per cent. The ages were for the most part between 10 and 20 years. All of the appendices containing the oxyurid had a hyperplasia of the reticulum cells of the lymph follicles of the submucosa which caused pressure from without inward on the mucosa thereby causing pain. In one of these appendices I found the oxyuris in the wall as well as in the lumen, in the others I found oxyurids in the lumen only." These were all chronic appendices and there is nothing in the pathological findings, Dr. Goodale tells me, to indicate that the oxyuris is any more of an irritant or menace than is the soft fecal matter so often found in the lumen.

There is another type of chronic appendix called the rigid appendix. In this there is an infiltration of the walls with fibrous tissue which destroys or interferes with the muscular action so that emptying is retarded or stopped even though the lumen is not changed from the normal.

This is not so serious a menace as the others but still is a menace, infection taking place from stagnation. In a certain number of these the fibrosis goes on to obliteration and there is nothing left but a fibrous cord. These are harmless as a rule but may be serious, as I found in a rare and interesting case of my own a short time ago.

During the night the patient, a man about 60, was taken with pretty severe abdominal pain centering around the umbilicus. The pain was somewhat colicky in character, but never entirely absent. He vomited once or twice. His wife gave him an enema with good result but no relief from the pain. I was not called till morning. I found him with a pretty severe steady pain in the right lower abdomen, pulse and temperature slightly elevated. The abdomen was somewhat distended and there was a large area of tenderness involving the whole right lower quadrant.

I felt that there was a definite surgical condition, probably an appendix, sent him to the hospital and operated. On opening the peritoneum a large amount of serous fluid was expelled and a loop of very dark-colored, evidently strangulated small intestine presented. The cause of the strangulation I found to be a fibrous cord surrounding the afferent and efferent gut which made up the loop. The loop comprised about twelve inches of gut. Upon investigation I found the cord to be the fibrosed appendix with its tip firmly adherent to the cecum about one half inch from its base. The appendix was removed and the patient made a good recovery.

We are told by the collectors of statistics that the mortality from appendicitis is increasing. At least one writer, Dr. Roy D. McClure, in a very excellent article in the August, 1931 *Annals of Surgery*, suggests that this may be due to the fact that the chronic appendices are not recognized and removed while still in the chronic stage. Many of them become acute and we know that the mortality from operation on acute appendicitis is much higher than on chronic. He does not argue from this, however, that we should go back to the old way of removing all appendices on suspicion. It is my impression that the only way this could be justified would be to show that the mortality from this operation plus the mortality from subsequent operations on the same patients for the real lesions, was less than the difference between the former and the present mortality from appendicitis. Even if this could be done it would leave out of consideration the unnecessary suffering of the patients subjected to a useless operation.

The reasonable solution is to make full use of all our present knowledge of symptomatology and physical examination, and of our laboratory and mechanical facilities in an effort to make an accurate preoperative diagnosis; and to study our abdominal cases with the idea of gaining more knowledge and developing present methods or finding new ones which will be helpful in the future.

An interesting side light, to me at least, on the supposed increase in mortality from appendicitis is furnished by figures compiled by Dr. W. E. Denning from the records of the Worcester City Hospital. He covers all cases during the fifteen years from 1914 to 1929, a total of 3730, and divides them into five-year periods.

This includes 288 cases with diffuse peritonitis in which of course the mortality was very high; 31.9 per cent.

The total mortality for the whole series was 3.9 per cent.

For the first five-year period the mortality was 4.6 per cent.

For the second five-year period the mortality was 4.05 per cent.

For the third five-year period the mortality was 3.2 per cent.

This shows a material decrease in mortality for each five-year period. From the same report I find 1345 chronic cases with a mortality of .74 per cent, while 2385 acute cases have a mortality of 5.6 per cent.

I still feel that careful questioning and physical examination of an intelligent patient will give us our most useful information. Our greatest difficulty in this is a proper evaluation of the psychic and nervous elements. Remembering our previous failures, where at operation we found nothing to account for the symptoms, we are inclined, I think, to attribute too much to these factors. There may be such a thing as imaginary pain and tenderness, and there may be such a thing as so-called functional disturbance of the colon or neurogenic colon, but those diagnoses must be made with great hesitation, and only after a most painstaking examination.

If we have a patient giving a history of repeated attacks of pain and soreness in the right lower quadrant, even though they be very slight and not at all disabling, if he has had one attack with vomiting and disabling pain and soreness, I think we will agree that his appendix should be removed. We can make the diagnosis in these cases from the history and physical examination alone. In my own experience only a few patients give such a definite history and the diagnosis must more often be arrived at, if at all, by a process of elimination.

The more common conditions from which chronic appendicitis must be differentiated are gall-bladder disease, duodenal or gastric ulcer, interference with urinary flow from the right kidney, mesenteric or retroperitoneal adenitis, adhesions, salpingitis or ovaritis.

The association of gall bladder disease, and of duodenal or gastric ulcer, with chronic appendicitis is so common as to give rise to a suspicion that there may be a causal relation.

Probably our most valuable accessory in diagnosis is the x-ray. A few years ago I did not feel that this was so, for one roentgenologist diagnosed a chronic appendix if it filled with barium, and another if it did not. A great deal of study has been given to this, however, and a great deal of progress made. A most informing article as to what can and cannot be done with the x-ray in helping to diagnose appendicitis is one by Joseph W. Larrimore of St. Louis in the December, 1930 number of *Surgery, Gynecology and Obstetrics*. He is very conservative and warns against making the diagnosis on roentgenological evidence alone. He does show, however, that the x-ray can give us positive evidence in a fair percentage of cases. If the x-ray shows a rigid appendix, or a fixed appendix, or concretions, or a definite fixed kink, or a narrowing of the lumen or a retrocecal location, we have positive evidence to add to our

clinical findings that there is a chronic appendicitis. We have in other words evidence that the normal emptying of the lumen is interfered with and as I have said previously, such an appendix is a menace and should be removed.

Some roentgenologists still feel that persistent failure of the appendix to fill, or retention of barium in the appendix for more than a given time is evidence of disease. Dr. Larrimore does not think failure to fill should be given much weight. Retention of barium after the cecum has emptied, and particularly after the cecum and right colon have emptied, he thinks is evidence of impaired motility.

In the physical examination, tenderness over the appendix region is considered a valuable sign. So it is, if the appendix happens to be in the so-called appendix region.

In about 13 per cent of patients the appendix is somewhere else than at McBurney's point. It is found under the liver, low down in the pelvis in the left iliac fossa and at all points between. Also the pain from a normally-placed appendix may be referred to any part of the abdomen.

In the x-ray we have a means of applying this test accurately. When you can see the appendix under the fluoroscope and see yourself actually palpating it, there is no chance for doubt as to whether the tenderness is in the appendix itself. This procedure should be a part of the routine of every x-ray examination for chronic appendicitis.

I feel then that any doubtful case with symptoms enough to demand serious attention should have the benefit of an x-ray examination. We will not get so much help as in gall-bladders or stomachs but certainly enough to make it worth while. I even feel inclined to say that the neglect of this is a culpable neglect.

Cholecystography is so highly developed that there is no excuse for confusing gall-bladder disease and appendicitis, and the same may be said about duodenal and gastric ulcers. The urologist should be able to save us from removing the appendix for ureteral obstruction, though I have had one case where I did just that on his advice. He demonstrated a kink in the ureter which he felt was due to a pulling of adhesions. Removal of a chronic appendix and freeing of adhesions cured the patient.

After we have used all our best efforts and secured all possible help, there will still be undiagnosed cases. Assuming that the symptoms are serious enough to demand relief there is nothing left but an exploratory operation. Let me emphasize again that this is justifiable only as a last resort.

There are a certain number of cases where we remove an undoubtedly pathological appendix and symptoms still persist. It is evident of course, that there must be some other lesion. It may be that we have failed to remove bands, congenital or acquired, which are pulling on the cecum and which, if present, probably were the original cause of the pain. The other lesion may be any one of the conditions which are so difficult to differentiate from the appendix.

We have not done our whole duty then when we have removed the appendix. Without thorough exploration of the whole abdomen in every case, we make ourselves liable to disappointing results. Theoretically this is right. We know however, that a thorough exploration of the stomach, duodenum and gall-bladder means a visual one, and that means an inconveniently high or a very long incision. It is a great temptation to most of us to content ourselves with a palpation of these organs, but we know that this will reveal only the grossest lesions and that we have not done our whole duty. As a practical thing it is probably justifiable to omit the visual examination of these organs if the x-ray examination of them has been satisfactory and negative and the appendix is definitely diseased. It certainly should never be omitted under any other conditions and even under these we lay ourselves liable to the same margin of error that occurs in the roentgenological examination.

I have refrained from debating the question as to whether there is such a thing as chronic appendicitis because it seems like a waste of words. I have quoted figures to show that in one large series of cases the mortality has progressively decreased, which is contrary to the reported general trend. I have tried to show the danger of neglected chronic appendicitis. I have called attention to the difficulty of making an accurate diagnosis and the necessity of making every possible effort to effect one, and have enlarged upon the value of the x-ray as a help. When we have been reduced to the court of last resort, the exploratory operation, I have pointed out the necessity for thoroughness in that.

As I said in my introduction, there is nothing new in any of this, but there is often much to be gained by reminding ourselves in detail of things which are so familiar that they have become relegated to our subconscious pigeonholes.

Discussion

Chairman Miller: This most excellent presentation brings up for further discussion a matter of supreme importance. I do not know anything which really arouses my own interest so much as this. I have been operated on for an acute appendix, and in regard to my own 8-year-old boy I am trying to decide, whether he should not perhaps have a prophylactic appendectomy done. I feel strongly about the danger of this particular disease, and I know I am going to gain light and get advice from this discussion.

The next speaker who will carry on this subject is Dr. Ralph W. French, surgeon at the Truesdale Hospital in Fall River.

Dr. Ralph W. French, Fall River: *Mr. Chairman*—I have not quite so much faith in the diagnostic value of x-ray studies of chronic appendix cases as Dr. Watkins seems to have. Positive findings, I think, are very helpful and often conclusive; but negative findings certainly do not rule out the presence of a pathological appendix.

Dr. Watkins has called attention to the possible causal relation between peptic ulcer, cholecystitis, and chronic appendicitis. I should like to enlarge upon that point. The alimentary abdominal organs are coördinated in their function. Disease in one part of the tract, to a more or less degree, disturbs the function of other parts. In a fairly large majority of cases of cholecystitis and peptic ulcer needing operation, we also find evidence of chronic appendicitis. Not infrequently we see cases of peptic ulcer and chronic cholecystitis which have been operated upon several years before for chronic appendicitis. This brings up the question of sequence of infection. The appendix has a fair amount of lymphoid tissue about it, which, I believe, like the tonsil, can harbor infection and in later life lead to upper abdominal disease.

Chairman Miller: I am very pleased to be able to call on a man who, in my opinion, is extremely skillful and learned in the diagnosis of diseases of the alimentary tract, Dr. Chester M. Jones, a visiting physician of the Massachusetts General Hospital.

Dr. Chester M. Jones, Boston: *Mr. Chairman*—The medical man in a discussion of this sort is more or less on the defensive, and Dr. Watkins has already made it impossible to offer any really adverse criticism. I think I should like to lay emphasis on certain things he has already indicated.

It seems to me there is no question that the important thing under discussion is the diagnosis of the condition. I must admit that a few years ago I felt a good deal of doubt about the existence of chronic or intercurrent appendiceal infection. I do not like the term chronic appendicitis, but I do not propose to take any issue with Dr. Watkins because his description coincides with my idea of recurrent appendicitis. It does seem to me of the utmost importance, however, that the emphasis be laid upon the accurate diagnosis of the condition.

I still see almost every week a patient carrying one, two or three abdominal scars. The first one is almost sure to be that of a so-called chronic appendix, and I still feel confident that it is necessary to insist upon careful study before an operation is decided upon. Dr. Watkins has already done that, and I should merely like to stress that from the point of view of the medical man.

Now, then, as to the means of diagnosis, I am sure that the physical examinations in many instances are rather disappointing. I agree with Dr. French that in most instances the x-ray examination is also frequently disappointing. That is often due to the fact, I believe, that the x-ray diagnosis is not well done. Where a thorough x-ray examination is done which involves a study of the patient not only by plates but by fluoroscopy at repeated intervals during 24 hours, and possibly 72 hours, then it is possible in many instances to get very definite aid. It is very rarely the case, however, that patients have their appendices removed because of a diagnosis of chronic appendicitis made through that procedure. It is expensive and takes time, but it is distinctly worthwhile.

The history also is most important; and I should like to mention, in passing, that we must at least consider what causes pain in the right lower quadrant. That has already been pointed out to some extent. I think I should like to lay a little more stress on constipation. A full cecum will cause pain at times and will be associated with extreme tenderness.

In my own mind there is no doubt whatever that there is such a condition as a spasticity of the colon. Down on the left side rather than on the right, that is, the sigmoid and descending colon may be extremely spastic and tender; but at times the cecum is just as tender, and the diagnosis of appendicitis at that time may be very difficult.

I know at the Massachusetts General Hospital we still talk about a nurse's appendix, and it has always been disturbing at times to operate for an apparent attack of appendicitis and find an absolutely normal appendix.

Dr. Watkins has mentioned gall-bladder disease and certain other conditions in the differential diagnosis, and I should like to add one or two others which are more rare but which also come into consideration. Especially in industry today we have many cases of lead poisoning which are clinically similar to chronic appendicitis. Tabes is another. Herpes zoster may be another, simulating a recurring acute attack.

So far as the production of pain is concerned, anything that disturbs the cecum will mimic the condition of appendicitis. We were able to prove that rather readily a year or so ago by passing balloons down into the cecum and then distending them. We were able to produce not only pain over McBurney's point but also the epigastric distress and nausea, characteristic of appendiceal inflammation, so that I think one has got to emphasize the mechanics of the situation and also the fact that anything disturbing the cecum may mimic the diagnosis of appendicitis. If, however, we have sufficient

evidence that the appendix is involved, of course it should be removed.

There are just two things I should like to say in closing. One is that there are certain cases, it seems to me, where removal of the appendix should be seriously considered in the absence of absolutely conclusive proof of the diagnosis. In diabetes, possibly in arthritis, and possibly in the presence of a supposed peptic ulcer, if we have fairly conclusive evidence of an appendiceal infection, I think at least the removal of the appendix should be seriously considered, although after study it may be possible to postpone the operation. Under such circumstances the removal of focal infection may be of real importance.

In closing, I should like to say that there certainly is no hurry in taking out a chronic appendix. I mean, by that, that when a patient presents himself in your office or at the hospital today, there is obviously no necessity of removing the appendix tomorrow for chronic appendicitis. If there is local tenderness, fever and leukocytosis the diagnosis is absolutely obvious, and the diagnosis is not chronic appendicitis but acute appendicitis. Otherwise, there is no hurry. In the second place, where it is difficult to make diagnosis, remembering there is no hurry it seems to me justifiable to demand not only that an immediate complete study be made, but also that the patient be seen in an attack. If the patient is seen in an attack, having had a history of attacks in the past, then the diagnosis has a fairer chance of being established, because in the attack there will be more signs pointing to the right lower quadrant, there probably will be an elevation of temperature, and there will be leukocytosis and a rise in the polynuclear count as well. If these findings are made I think one has a right to remove the appendix.

CHAIRMAN MILLER: I am going to take the liberty of asking for a few words from a gentleman who has not the slightest intention, I presume, of saying anything. I see the Roentgenologist of the Springfield Hospital, Dr. Van Allen, and I am going to ask him if he will say just a word about what he thinks of diagnosis in appendicitis.

DR. HARVEY W. VAN ALLEN, Springfield: *Mr. Chairman*—I am certainly surprised to be called on to speak in a surgical meeting. In regard to the x-ray, I think, with one of the speakers, if fluoroscopic examination is made we can do a good deal for the surgeon in regard to a diagnosis of appendicitis. Whether a tender point in the right abdomen is an inflamed appendix can best be determined by pressure with the finger during fluoroscopic examination. In this way when the visualized appendix corresponds to the painful point, even though the location changes with the pressure, a diagnosis can be made. The plate, of course, gives us an idea as to whether the appendix empties. There is much discussion as to the reliability of this sign. Alone it is of small value, but with other findings it is of great importance.

I think that a good many times we should be helped by the surgeon a little more. The case is sent in as a gastro-intestinal tract case, and if we carry that case through and do a thorough stomach examination and then follow through the whole bowel and watch the appendix, it makes a 72 hour examination many times and produces a good deal of x-ray exposure for the patient. So, if the surgeon could help us by saying that he believed ulcer of the stomach was present, or ulcer of the duodenum was not present, that would be extremely helpful.

So my plea would be a little better coöperation with the surgeon. He can start us off with a little better idea of the case than by simply turning it in as a gastro-intestinal tract case. You will find that the roentgenologists are very willing and anxious to help the surgeons in their difficult tasks.

CHAIRMAN MILLER: This paper is now open for general discussion. We will be pleased to hear from anybody. Dr. Young, will you kindly come up here. Dr. Edward Young, surgeon at the Massachusetts General Hospital of Boston; surgeon-in-chief at the Faulkner Hospital.

DR. EDWARD L. YOUNG, Boston: *Mr. Chairman*—I should like to have a word in this chronic discussion. First, I should like to say that I consider Dr. Watkins' paper one of the sanest papers on chronic appendicitis that I have heard in a long time.

You may remember that a distinguished clinician several years ago said there were two kinds of appendicitis, "acute appendicitis and appendicitis for revenue only". I think we do not entirely agree with that, but there is too much truth in it entirely to disregard so that we want to take the diagnosis of chronic appendicitis very seriously.

I am sure there is no such thing as a primary chronic appendix. It is true that there is such a disease, but the condition is always secondary either to repeated mild attacks or to some other condition. In making the diagnosis, there are over forty different diseases to be considered and because of the atypicality of appendicitis, none of them can be disregarded.

There is one condition, however, that I want to speak of, which is not well understood, which needs the combined study of an x-ray man, a surgeon, and a good medical orthopedist. I have seen a good many of them with Dr. Loring Swaim, generally in that type of individual that we call the ptotic. For lack of a better name, I have called it cecal stasis and it is due primarily to a poorly acting cecum which does not empty well and because of fecal retention, starts up a

chronic irritation. This causes a few adhesions which become crippling and the chain of symptoms begins. Dr. Rae Smith of Los Angeles has written about it and has emphasized the lateral adhesions which tend to constrict the ascending colon. In this type of case the symptoms are in the right lower quadrant. It is one of the very common types of chronic appendicitis, which removal of the appendix alone will not cure. The patient must have, first an accurate diagnosis; this means an understanding of the condition by the surgeon, the x-ray man, and the medical man. There must be a competent attempt by means of exercises, supports, and positions to mobilize the ribs so as to increase room in the upper abdomen which will allow organs to stay up when they are lifted up. After six months or a year of this medical orthopedic treatment, and only then, should surgery be undertaken. The operation consists in freeing adhesions, (and in a certain number of cases that is all that is essential), and if necessary, plicating the cecum so that it will have a narrowed lumen and will rest in the iliac fossa and not in the true pelvis. I have done about thirty of these cases in the last few years, always with benefit, though it must be remembered that it is the type of case where the ptosis alone will cause certain symptoms. I have operated on two patients who had their appendices out previously without improvement who were cured after the complete operation.

I think what Dr. Van Allen said is of very great importance: there must be intimate coöperation between the x-ray man and the surgeon. Any x-ray man that will send you back a diagnosis of chronic appendicitis should have his report thrown in the wastebasket. Go to someone who will talk the same language, will tell you the conditions, and then you will together get an accurate diagnosis.

CHAIRMAN MILLER: Dr. Whitaker, will you come up here. Dr. Lester Whitaker, of Boston, surgeon at the Massachusetts Memorial Hospitals.

DR. LESTER R. WHITAKER, Boston: For years it has been assumed that a direct causal relationship exists between gall-bladder disease, peptic ulcer, and appendicitis; but, so far as I am aware, this has never been proved, and I doubt that it exists.

CHAIRMAN MILLER: Is there anyone else who has anything to offer? Has anybody anything further to add to what has been said? If not, Dr. Watkins, I will ask you to close the discussion.

DR. WATKINS: *Mr. Chairman*—I just want to say one word about the x-ray examination of these abdomens. Until within a short time, a year or perhaps a little more, I was absolutely skeptical about the value of x-ray in these conditions. I felt that I got no help at all. Since then I have been converted, and I feel exactly as I expressed it in my paper.

CHAIRMAN MILLER: The saving of life in this disease of acute appendicitis must to a certain extent come back to the medical man, to the general practitioner, and I am very pleased to be able to present to you now a medical man who will give you his views on this subject. Dr. George Steele is a visiting physician at the Springfield Hospital. He will talk to you on "Medical Aspects of Appendicitis." Dr. Steele.

MEDICAL ASPECTS OF APPENDICITIS*

BY GEORGE L. STEELE, M.D.†

WE are inclined to think of appendicitis as a condition coincident with the improved living conditions of the past few generations. There is, on the contrary, evidence that the disease was recognized as an abdominal inflammation 500 years ago and from time to time during the past five centuries an occasional contribution to the literature has been made regarding it.[1] It has been called by various names and inflammation of the bowels and perityphlitis are terms which were in common use half a century ago, just preceding the term appendicitis.

In 1370 John of Arderne, the earliest of English surgeons, wrote treatises on the subject, discussing it as passio iliaca. He was a well-educated man who got his training by an adventurous career as an army surgeon in the Hundred Years War. His surgical writings were widely used in England for nearly 200 years. Three hundred and forty-one years later, in 1711, Lorenz Heister, a German, made the first postmortem section of appendicitis, and comparatively soon after that the first operation for localized appendicitis was reported. This was done by Mestivier in 1759. He found the pathologic appearances clearly described by Heister 48 years previously. Yet these landmarks left no impression on practise. James Parkinson, one of John Hunter's pupils, in 1812 reported the first case of appendicitis in English, being also the first case in which perforation of the appendix was recognized as the cause of death[2]. His description follows:

"Upon examination, the whole surface of the peritoneum was found inflamed, and covered with a thin coat of coagulable lymph; and slight

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adhesion had taken place between the peritoneum covering the viscera, and the parietes of the abdomen. The viscera, independent of the inflammation of their peritoneal covering, appeared in a perfectly healthy state, excepting the appendix vermiformis of the coecum. No diseased appearance was seen in this part near to the cecum; but about an inch of its extremity was considerably enlarged and thickened, its internal surface ulcerated, and an opening from ulceration which would have admitted a crow-quill, was found at the commencement of the diseased part, about the middle of the appendix, through which it appeared that a thin, dark-coloured, and highly fetid fluid, had escaped into the cavity of the abdomen.

"Upon opening the appendix, a piece of hardened feces was found impacted in that part of it which lay between the opening, and that portion of the appendix, which was not evidently marked by disease."

Willard Parker of Francistown, N. Y., operated for appendicitis in 1864 and in 1886 the late Reginald H. Fitz gave conclusive demonstrations of the pathology of perforating inflammation of the vermiform appendix.

Appendicitis was for years treated medically with ice, enemas, and morphine. It is within the memory of surgeons still active that it was considered proper to wait until an appendiceal abscess formed and drain it. With the advent of aseptic surgery, abdominal operations became common and surgical skill and technique made great advances and the death-rate undoubtedly was lowered. Appendicitis soon ceased to be regarded as a medical condition, but the situation is still far from satisfactory.

In 1930 there were 606 deaths from appendicitis in Massachusetts (a rate of 14.3 per 100,000 of population or a distribution rate of 1.2%)[3]. This rate has varied but a fraction of 1% during the past five years. The death-rate in many other surgical conditions has shown a decrease in the past decade, but appendicitis still claims as many victims as five or ten years ago.

Surgical technique and postoperative care have improved so much that it seems unlikely that the surgeon can do much more to lessen the mortality of appendicitis, unless it be closer control of the occasional operator. It appears that the burden must for the most part be shouldered by the medical man and the patient.

Rarely is a physician called before the patient has been ill for at least two hours and often eight or ten hours have elapsed. After diagnosis has been made it is usually another two hours before operation can be performed. It is during this period that the inflamed appendix may go on to perforation, or gangrene and peritonitis occur. This time must be shortened by education of the public and by insistence on the part of the attending physician that immediate operation be performed as soon as the diagnosis is made. Purges must not be given. Lord Moynihan[4] states that he has never seen perforation of the appendix that was not provoked by purgation and that if aperients were never administered there would be few if any deaths from this disease. The symptoms should not be masked by morphine or ice until a positive diagnosis has been made. Deaths in appendicitis are due to complications and not to the primary disease.

Acute abdominal pain which persists for more than an hour or two is generally indicative of a surgical condition. The appreciation of this rule by the laity and its application by the physician should be of tremendous value in bringing the patient to the surgeon before complications have progressed. Exceptions to this rule are medical conditions such as abdominal pain occasionally seen in diabetic acidosis, coronary thrombosis, and the referred pain of lobar pneumonia. Surgical conditions which necessitate differential diagnosis are not within the sphere of this paper.

It is the family doctor who is generally summoned for abdominal pain and on him rests the responsibility and the satisfaction of early diagnosis, and on his alertness depends whether a diseased appendix is removed before complications have occurred. Confirmation of the diagnosis and support of the surgeon should, if possible, be awaited until the patient has reached the hospital, rather than incurring further delay at the patient's home. The patient can be prepared and necessary laboratory examinations made if the surgeon is not at once available.

It has been mentioned that certain medical conditions may confuse the picture and make the decision more difficult. Important among these is the abdominal pain frequently found in acidosis accompanying diabetes. This pain closely simulates appendicitis and requires careful consideration. It may be general over the abdomen or localized in the right side. The leucocyte count is similar to that found in appendicitis. Vomiting, tenderness, and muscle spasm may be present. If there is a diabetic history, this condition should be kept in mind and operation postponed until the acidosis has been overcome. The condition should be treated as a severe diabetic acidosis; glucose and saline should be given subcutaneously or intravenously and insulin given frequently in adequate doses and close laboratory control maintained. It must not be forgotten that there may be a persistence of high blood sugar and the presence of acetone in the blood, after urine examinations have ceased to show them; and symptoms may continue until these are corrected. The acidosis may be overcome in a few hours. If the abdominal symptoms are caused by diabetes they will disappear as the acidosis is cleared. If a definite diagnosis of appendicitis is made in a diabetic patient, our present-day management of the condition renders operation comparatively safe

whether the anesthesia be general or local. The outcome greatly depends on the preoperative and postoperative care by the physician.

It has long been recognized that referred abdominal pain simulating appendicitis not infrequently is caused by lobar pneumonia involving the lower lobe of the right lung. Careful weighing of the clinical symptoms, and competent examination of the lungs may still leave the diagnosis in doubt. Rigidity of the abdomen is present and the temperature is elevated. If the ratio of the pulse and respiration is reduced to three or two to one, there is an increased liability of the lesion being above the diaphragm. X-ray of the lungs will clear the situation. In acute pleurisy and in pleural effusion of the right chest, the pain is not infrequently reflected to the right side of the abdomen and motion of the chest may increase the abdominal pain. In such cases shallow respiration, pleural friction rub, and changes in breath and voice sounds should make the diagnosis. If the appendix of a patient ill with a respiratory condition must be removed, avertin and local anesthesia are preferable to general anesthesia.

Coronary artery disease is at times most misleading as the symptoms may be chiefly abdominal. The sudden, severe onset, prostration and dyspnea in a patient of middle life or over should put the examiner on his guard. The history of previous attacks of angina pectoris may be elicited. Râles may be heard at the bases of the lungs or some definite cardiac signs such as feeble heart sounds or fibrillation, premature beats or a pericardial friction rub may be found.

Acute gastro-enteritis from eating unwisely or too heartily at times appears to be a forerunner of acute appendicitis and must always be considered carefully and with respect. Diarrhea with frequent foul stools, widespread tenderness over the abdomen, some degree of distention, cramps relieved by intestinal evacuation, and low fever with the history of dietetic indiscretion may serve to differentiate these conditions and acute appendicitis. If the fever rises or the tenderness becomes localized in the right side of the abdomen, appendicitis must be diagnosed[2].

There are other medical conditions such as lead colic, tabes, and ulcer of the stomach which must be considered in diagnosing the atypical case of appendicitis.

The diagnosis in the presence of the classical symptoms is readily made by taking a careful correct sequential history. It is the unusual case which requires sound judgment. Early in the attack, tenderness, muscle spasm, leucocytosis and constitutional symptoms may be absent and abdominal pain may be present as the only symptom. The pain may be only moderate with periods of complete or partial relief yet severe enough to interfere with sleep and to cause restlessness. Rectal examination, so frequently forgotten, may disclose tenderness low in the right side of the abdomen. Later, localized tenderness and other characteristic symptoms appear. A feature rarely absent is failure of the patient to eat after the onset of pain. When the neglected inflamed appendix has gone on to gangrene, and pain and temperature have abated, there is a period without any marked symptoms. A lull before the storm! The history of severe abdominal cramps coming in waves, fever and accelerated pulse followed by a partial cessation of symptoms should warn the physician against temporizing. This is illustrated in the following case:

A patient seen after a twelve-hour illness gave the following history. He had awakened at the usual time and did not feel like eating his breakfast. His stomach felt unsettled so he took a saline purge and went to work. Two hours later abdominal cramps began and became so severe that he returned home. The pains were attributed to the purge and he was given an opiate. Waves of cramps with periods of comparative comfort continued until evening when he was found to have an elevated temperature and pulse rate, muscle spasm, and localized tenderness over the right abdomen. Operation disclosed a perforated appendix and quantities of free pus in the abdomen.

Chronic appendicitis is a diagnosis requiring considerable thought. It should only be made after painstaking examinations or humiliation may result when at operation some other condition is discovered such as ulcer of the stomach, visceroptosis, or tuberculous glands in the ilio-cecal angle.

The death-rate[5] in acute appendicitis with its complications has long stood higher than ten per cent and the outstanding cause of the high death-rate is failure to make the correct diagnosis within the first twelve to eighteen hours.

Appendicitis in children presents a problem in which education of the parent is most necessary to lessen the mortality[6]. Older children can describe pain and other subjective symptoms dependably. In younger ones this aid is lacking, consequently appendicitis frequently proceeds to perforation with peritonitis or abscess formation before the true condition is suspected.

I believe it is the feeling among parents that intestinal upsets with colic in children are generally the result of errors in diet or constipation, and cathartics are freely given before the doctor is called. This may have much to do with furthering what might otherwise be a simple pathologic process.

Any child with acute abdominal pain, even slight tenderness over the lower abdomen, a moderate fever and leucocytosis should be considered to have acute appendicitis unless other definite pathology can be found. The mortality rate is much higher in this disease than in adults and is given as 14.4% by the Bureau of

Vital Statistics[8]. Strange to say, the rate has been rising in this country while reports from other countries where vital statistics are available show the death rate to be either stationary or gradually falling.

Early diagnosis[9] and immediate operation are the counsels of perfection, but failure to diagnose early is by no means always the fault of the practitioner, for in children and old people the diagnosis may be difficult and many persons regard a stomach-ache too lightly and do not send for the doctor until complications have arisen.

Appendicitis remains the most common abdominal emergency which the medical man is called to attend and shares the dangers incident to delay with perforation of viscera, intestinal obstruction and tubal pregnancy. More has been accomplished in lessening the mortality in the past fifty years than in the preceding five hundred. The diagnosis should rarely be missed if earnest intelligent effort is made by the examiner and if the physician and laity will keep in mind that acute abdominal pain of more than an hour or two in duration is generally indicative of a surgical condition.

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Discussion

Chairman Miller: Dr. Steele, we as a surgical group want to express to you our very sincere and deep appreciation for all the time and effort which you have devoted to the preparation and presentation of this most splendid paper.

Dr. Steele: Thank you, sir.

Chairman Miller: You mentioned the supremely important work of the late Dr. Reginald Fitz. It has always been a matter of satisfaction and pleasant memory to me that, at Dr. Fitz's last clinic given in the Harvard Medical School, I was the last man he called on. I was ordered to come down front and was quizzed by Dr. Fitz on the day he retired from teaching.

We are particularly gratified to have here one of our very best medical men in Boston and the son of the late Dr. Fitz, and I am most pleased to present to you Dr. Reginald Fitz, visiting physician at the Peter Bent Brigham Hospital.

Dr. Reginald Fitz, Boston: *Mr. Chairman and Members of the Society*—I find it extremely difficult to try to discuss Dr. Watkins' paper, as it has covered the subject so perfectly. Recently, I heard a most interesting paper by Dr. Bower of Philadelphia on appendicitis and he has been kind enough to send me some of his lantern slides with which to illustrate certain of the salient points brought out by Dr. Watkins. (Lantern demonstration.) In summary, these slides show that appendicitis, on the whole, is a disease of young people; that peritonitis is the chief cause of death; that delay in operation increases the mortality very remarkably; and that the medical treatment of appendicitis with laxatives is pernicious.

It so happens that, 41 years ago at this very time and before this very Society, the question of the diagnosis and treatment of inflammation of the appendix was under discussion The discussion, then, was carried on by various Fellows whose names we shall always be glad to remember with pride—Fellows like Dr. J. C. Warren, David W. Cheever, R. H. Fitz, John Homans, C. B. Porter, J. W. Elliot and F. C. Shattuck. There was one young medical firebrand present, practicing at Waltham at that time, by the name of Dr. Alfred Worcester. What he had to say then seems still to epitomize the medical aspects of appendicitis. Said Dr. Worcester, in that sincere, forceful way which we all so much admire:

> "Gentlemen, appendicitis must be treated by the surgeon from the first to the last; it is just as much a surgical disease, and entirely within his province, as any disease that can be thought of. There is no use in leaving the physician to decide when to call the surgeon in, for he will not call him in until he finds that he, himself, cannot save the patient. It is easier to diagnosticate the disease in the first 24 hours than at any subsequent time. All the symptoms are more apparent at first. I would rather have appendicitis under the care of a country surgeon who is willing to operate the moment he recognized the disease, than to be under any of the surgeons who would not operate, although they recognized the disease early, because it is impossible to tell what cases are going to the bad. The treatment of appendicitis belongs to the surgeon from the first; and in the great majority of cases the patient's chances will be better if he is operated on as soon as the diagnosis is made, and, that being so, the duty rests upon every one of us to operate just as soon as diagnosis is made."

Chairman Miller: Gentlemen, think of it! Forty-one years ago today that discussion was held in this same Section of this same Society, and that sturdy youth who has been quoted by

Dr. Fitz has been good enough to accept our invitation to come here at the end of 41 years and talk to us for a few moments again about appendicitis. It is a great honor and satisfaction to introduce again Dr. Alfred Worcester.

Dr. Alfred Worcester, Waltham: This is a very different reception from what I had 41 years ago! I was pounded heavily on that occasion. I have already been very much interested this morning and expect to be still more so before this session closes, for one reason because the same difference of opinion seems to exist regarding the pronunciation of the disease. I am reminded of a discussion between two boys who had suffered from it, one said from appendicitis and the other fellow said from "appendiceetis," but the first fellow said he had had his disease in the side and not in the seat.

This discussion about possible treatment previous to operation reminds me of a recent case where a Harvard student was operated upon in just two hours and thirty minutes after his first pain. He was a well boy on Sunday. He ate a good noonday meal and had no trouble until 4 o'clock that afternoon. He had been in college only a few days. At my opening talk to the Freshmen I have impressed upon them that they must call a doctor quickly if they have belly ache, and, remembering that, he called for a doctor at five o'clock, only one hour after he had his first pain. At half-past six his belly was opened, but the appendix had broken through. He nearly lost his life.

Well, to go back, beyond the memory of almost all who are now present, to what happened before the surgical treatment of appendicitis, it will hardly be possible to make you realize what we used to see. I had lost several cases of appendicitis in the old era before operation was thought of. I have seen the belly wall gangrenous before death. One of my medical brethren, long since dead, who had appendicitis when he was young had the abdomen burst and a gush of pus go over his bed. The first case that I had, in a splendid, strong man came nearly to that condition, and after his death, when I found his belly full of pus, I made up my mind that never again would I let a patient die so. I would make an antemortem examination anyway.

That was firmly in my mind when from the Massachusetts General Hospital was sent out to my care a little girl with a hopeless prognosis. She was sent out to die, as was the sweet, lovely way of the Massachusetts General Hospital 40 and 50 years ago—they had that much confidence in the general practitioner then. That was August 18, 1886. The diagnosis was septic psoas abscess, and as it was thought not safe to operate because of her high fever, the parents were advised to take the little girl home. They were told that as in a few days she surely must die, it would be better all round to have her die at home than in the hospital.

As I have said, I had made up my mind that never again would I let such a patient die without operating. Here was this child with terrific pain and exquisite tenderness in her right belly, but where was I to incise? I went in through the loin with an aspirating needle and got stinking pus. Following the needle with a bistoury and then with the old Bigelow dilators I opened the incision until I could get my fingers in. I told the surgeon assisting me that I had found a foreign body.

"What is it?" he asked.

"Well," I said, "it is corrugated, and it is certainly not living tissue."

He said, "You have gone into the cecum and have got hold of a piece of feces."

"No, it isn't that," I said.

"Pull it out, then," he said.

"I can't," I said, "it's hitched on."

Well, he gave me a double tenaculum, and I got hold of the thing. It did not come off very easily but did finally. That surgeon insisted it was feces; and, to my mortification, he reported it as such two or three years afterward. I did not know what the thing was. Fitz's paper had not then been published. I knew of the appendix vermiformis only in the form of an earthworm. I did not know it could be as big as a thumb and as nasty as that thing was. Not knowing what it was, I threw the stinking thing away, and so mortified was I that after Fitz's paper was published I did not dare mention the case for years afterwards.

I myself had had similar attacks where I had been laid up sometimes for a day with the pain, and finally when in 1887 I was stricken with the real thing and became so desperately ill that my life was despaired of, some of my Boston friends came out. Maurice Richardson came. Dr. Fitz also came, but as I never had seen him before except at autopsies, I looked at him somewhat askance. Fortunately for me, John W. Elliot was there, and, although Drs. Fitz and Maurice Richardson protested against the operation, I feebly begged for it. John W. Elliot knew that I had operated on the little girl who had been sent out from the Massachusetts. I said, "I am dying of the same thing that you sent that little girl out to die with, and she got well because I opened her belly and you have got to open mine if I am to live." I also said, "I know what you fellows mean. You mean to come out tomorrow and do a postmortem. Why can't you do an antemortem?" Jack Elliot said, "Do you really mean you want your belly opened?" I said, "Give me a knife's handle and I'll show you whether I do." John W. Elliot afterward said that was too much for him, and he did open the belly, and, if E. Amory Codman is right, I have the distinction of being the first man who was ever operated upon for appendicitis, that is, I am the first man whose belly was cut open by a surgeon to go after an abscess which had not pointed. Until that time it was considered prop-

er surgery to cut through the belly wall if only for opening an abscess distinctly adherent.

After a long and stormy convalescence, when I recovered, Boston surgeons introduced me handsomely. When I was on exhibition at the Royal College of Surgeons as one who had taken out an appendix myself and also as one who had survived the operation, I was told, in that magnificent British way, "We do not have appendicitis in England."

CHAIRMAN MILLER: Gentlemen, please rise and applaud.

CHAIRMAN MILLER: Dr. Worcester, the pleasure and satisfaction which you have given us are really beyond my power of expression. We thank you most heartily.

DR. GEORGE A. MOORE, Brockton: *Mr. Chairman and Members of the Section*—I think our Chairman should be congratulated in his choice of a subject which is of such vital interest to so many of us at this time. I question if my own experience regarding appendicitis differs greatly from that of many other men in this room. In my training at the Massachusetts General Hospital and my early years of practice I grew up in the belief that appendicitis was one of the simple, elementary surgical diseases, and I entertained that idea for several years. In studying the statistical reports that were published I realized that not only was the mortality of appendicitis increasing but that my own mortality of cases was too high. I felt probably as many other men have that it was necessary to revise our methods of treatment if possible.

Dr. Steele's paper has been of much interest to me because so few men in recent years have discussed this problem from the medical point of view. There is little I can say regarding the medical complications because he has covered the field so thoroughly. I can only emphasize some of the complications that he has touched upon.

There is a question in my mind which I had hoped Dr. Worcester might discuss regarding the twentieth century appendix. Is it different in its physiology and pathology from the appendix of the nineteenth century? We all know that our standards of living have improved greatly; that the roughage has been removed from our food and that we are consuming a great deal more sugar than we used to. Has our intestinal flora also changed?

We also hear a great deal about the increase in the incidence of appendicitis. I have found nothing in the literature to show statistically that appendicitis is on the increase. Probably it is but nobody has brought this out in the literature.

In regard to the medical study of these cases before operation, I have a rather firm conviction that few cases, a comparatively small percentage are studied carefully by competent internists before operation. In some of the wards of our large hospitals, studies are carried out. Dr. King of the Massachusetts General has reported their studies of ward cases in regard to the incidence of massive collapse of the lung; but in probably 80 per cent of all cases of appendicitis, and I think it might be fair to state more than that, we call in the medical consultant, not to advise us how much surgery the patient will stand but to break the news to us that we have overestimated the patient's strength, and the heart or kidney or some other system has been overtaxed. That is, we have not studied these patients as we should. We have tried to fit the patient into the ideal operation rather than to fit the operation to the patient.

I should like to ask Dr. Steele if he has the same impression of such medical studies as I have.

I have gone over my private cases to determine if possible the number of medical complications which were recognized. I realized that probably many of them were not recognized. I have also attempted to determine in the cases that died if some of them could have been saved by more careful medical study.

(Discussion of slides.)

I think that is all I want to call your attention to in the way of medical complications, except to mention the three heart cases that died and two uremias to bring out the point I want to in regard to our own study. I think we definitely overestimated our patients and did not have careful medical study of these three heart cases. One of the uremias Dr. Fitz saw with me, the typical low functioning kidney with an appendix that required operation at once. It may be that we did too much surgery on him. If we had merely drained we might have saved him, but certainly he had everything that was coming to him in the attempt to make the kidneys function after operation.

The other uremic case is a question of operating on an empty bladder, which I think is something to be thought of by all men doing acute abdominal surgery.

My statistics are as follows:

APPENDICITIS

Mortality

Total Number of Cases	1277
Number of Deaths	22
Mortality	1.7%

Medical Complications

Acute Tonsillitis	3
Streptococcus Throat	4
Influenza	11
Acute Bronchitis	9
Pneumonia	3
Postop. Atelectasis	4
Pulmonary Tbc.	9
Measles	2
Scarlet Fever	2
Hypertension	12

Medical Complications

Heart—Myocarditis	6
Acute Nephritis	8
Uremia	3
Duodenal Ulcer	2
P. O. Ac. Dil. Stom.	6
Diabetes	8
Hyperthyroid	3

Causes of Death

	No. Cases
Diff. Peritonitis	10
Heart	3
Uremia	2
Septicemia	1
Scarlet Fever	1
Pulmonary Embolus	1
Intest. Obs.	3
Ac. Dil. Stomach	1

CHAIRMAN MILLER: We apparently have a substantial amount of time in which to finish; on the other hand, if we allow the discussion to go on much longer at this particular moment, we will be probably crowded at the end. I am therefore not going to call on anybody else to discuss this paper at this time; but later on, there may be occasion to return to it, and I will ask Dr. Steele if he will now close.

DR. G. L. STEELE: I have enjoyed the discussion, as you all have, and I am sure Dr. Fitz, Dr. Worcester, and Dr. Moore have added a great deal to our knowledge this morning.

Regarding medical studies in surgical cases, I can only speak for my own locality, and my feeling is that there is very good coöperation between the medical and the surgical groups in western Massachusetts, and that I think it is unusual for a patient to go to operation without a fair amount of medical study which at least includes the usual routine laboratory examination and a chest examination.

Dr. Worcester, I notice, still uses the long "i" in the pronunciation of "appendicitis".

CHAIRMAN MILLER: I am now very pleased to present the reader of the next paper, Dr. Howard M. Clute, of Boston, surgeon at the Lahey Clinic, whose subject is "Factors Reducing Mortality in Acute Appendicitis."

FACTORS REDUCING MORTALITY IN ACUTE APPENDICITIS*

BY HOWARD M. CLUTE, M.D.†

THE reduction of the mortality in acute appendicitis is dependent upon the early diagnosis of the serious type of this disease, a proper operative technique in patients having this condition and the adequate management of such complications as arise after operation. It is my desire to discuss in this paper these points, since I am convinced from our experience that the mortality in appendicitis is so largely related to these factors.

Everyone agrees that early diagnosis is probably the largest, single factor necessary for the avoidance of complicated cases of appendicitis and the reduction of mortality. The statement, however, that early diagnosis is essential has been so frequently repeated that it will no longer attract attention, unless we can emphasize some particular type of appendicitis in which the symptoms of the disease are present within a few hours of the onset, and in which the opportunity to save the patient from peritonitis is present but a relatively few hours after the first pain.

There is a type of appendicitis in which the pathology is fairly constant, the symptoms are quite characteristic and the mortality is extremely high. In fact it is our belief, that the great majority of the fatal cases in appendicitis arise in this particular type of the disease which can be recognized from the clinical history and which is based on a very definite pathology, fundamentally the same in each case. I am referring to an acute obstruction of the lumen of the appendix by either a fecolith or a band. This condition of appendicular obstruction was first described by Professor D. P. D. Wilkie of Edinburgh[1]. In this paper and in numerous other contributions since this time, Wilkie describes two general types of appendicitis, the obstructive and the inflammatory. Each of these is associated with definite clinical symptoms and typical pathological findings. Since our attention was directed to Wilkie's work we feel that we have been able to definitely reduce the mortality in those appendicitis patients whom we have seen by the recognition of the dangerous, obstructive type of appendicitis which he describes and of the pathology which accompanies it.

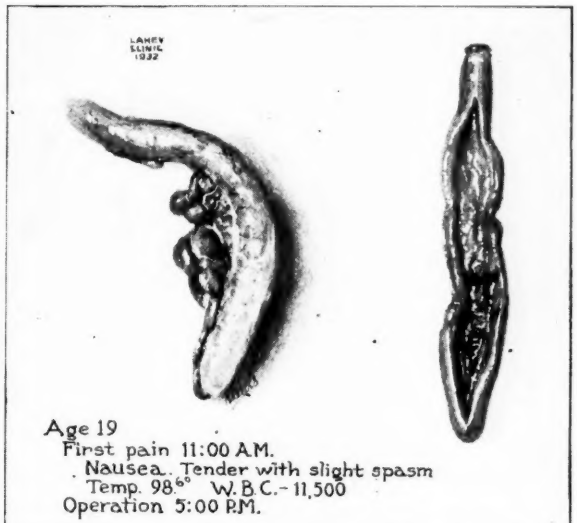

FIG. 1. Obstructive appendicitis—Gangrene of the distal third of the appendix following obstruction to the lumen with a fecolith, decomposition of the fecal contents beyond the obstruction. Beginning perforation of the wall seen in the right-hand drawing.

In the last five years we have operated upon four hundred and thirty-four cases of acute ap-

*Read before the Surgical Section of the Massachusetts Medical Society at its Annual Meeting, June 10, 1932.
From the Lahey Clinic, Boston.

†Clute—Surgeon, Lahey Clinic. For record and address of author see "This Week's Issue," page 379.

pendicitis in the Lahey Clinic. Of these patients nine have died, a mortality rate for the entire series of 2.07%. Eight of these nine cases had definite obstructive appendicitis and it is at once obvious that nearly 90% of our mortality can be readily traced to this particular lesion.

FIG. 2. Obstructive appendicitis with fecolith showing in the lumen of the appendix. Delay in removal of this type of appendix lesion is responsible for a large part of the mortality in appendicitis. Its symptoms are typical and suggestive of the obstructive basis of the pathology.

The pathology of this serious type of appendicitis is fundamentally dependent upon the presence of some obstruction in the lumen of the appendix. This, most commonly, is a fecolith although there may be a sharp kinking of the appendix by peritoneal bands or a stenosis of its lumen from previous mild, inflammatory attacks that finally cause the obstruction. When, following some increase in size of the fecolith, the obstruction suddenly becomes complete, decomposition of the fecal contents distal to the obstruction occurs. Gangrene of the wall rapidly develops from loss of blood supply and rupture of the appendix may occur within six to twenty hours from the onset of the first obstructive symptom.

In this closed loop obstruction, as Wilkie has repeatedly stated, pus is rapidly formed due to the decomposition of the fecal contents. There is, however, no absorption of note from this small collection of purulent material so that a febrile reaction does not occur; furthermore, there is no serious irritation of the peritoneal covering of the appendix in the early stages and, therefore, the omentum does not come near the area to wall off the appendix. As the tension inside the obstructed appendix increases, the amount of purulent material becomes greater and finally rupture of the appendix occurs and a rapidly spreading, diffuse peritonitis arises from it. This type of obstructive appendicitis results fatally because there has been no opportunity for the omentum and the intestine to wall off the infected area and feces tend to pour continually from the point of perforation.

The clinical symptoms which accompany obstructive appendicitis are as a rule quite typical of the lesion, but it is only by appreciation of the pathology of this very serious condition that we may detect its presence and so urge early operation within the first twelve to fifteen hours after the symptoms have started. The typical symptoms in these cases are the sudden onset of pain, which may waken the patient from sleep or may occur at any time in the course of his usual occupation. The pain is colicky in character, it lasts for a little while and then quiets down; it returns after a time with even more severity and may then be persistent. An early and definite elevation of the white count is often seen. The temperature is usually normal. There is relatively little localized tenderness or spasm and this, we feel, is most important to recognize since one may wait too long to detect the spasm and tenderness before urging operation in this type of case. In fact, it is our belief that the diagnosis of acute appendicular colic may well be made upon this typical history of pain in the lower abdomen, often referred to the region of the umbilicus without any more localizing symptomatology of the condition than tenderness over the appendix area. In the absence of evidence of ureteral

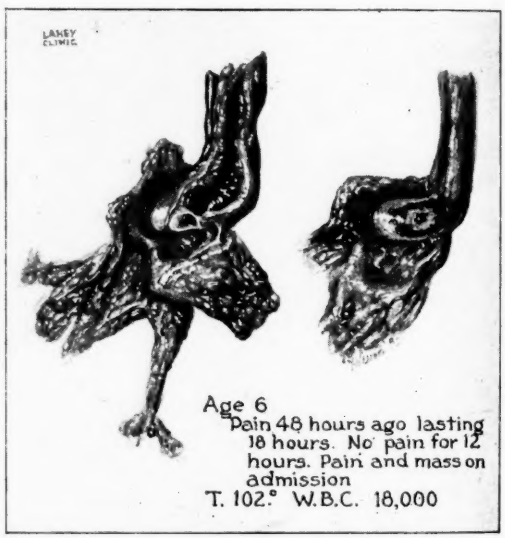

FIG. 3. Obstructive appendicitis with perforation of the appendix by the fecolith. Fortunately, in this case, the omentum was near at hand, walled off the abscess at the appendix tip and so prevented a generalized peritonitis. Usually in obstructive appendicitis omentum is not present at the point of perforation and a virulent peritonitis rapidly follows the perforation.

stones I should have no hesitation in recommending operation to such a patient.

A typical example of this type of obstructive appendicitis was seen a week ago in a surgeon in this city. He was awakened from his sleep at five o'clock in the morning with a severe pain in the right lower abdomen. It lasted for about fifteen minutes and was extremely severe in that interval. It then disappeared but returned in about an hour with less intensity. He was examined at ten o'clock, five hours after the onset of the pain. He showed definite tenderness over the appendix area; no muscle spasm was present. His temperature was normal, his white blood count was 15,600, and his urine was normal. A diagnosis of obstructive appendicitis was made and operation advised. His appendix was found to have a complete obstruction by a fecolith and the distal end of the appendix was covered with fibrin and filled with pus. Six hours had elapsed after the first pain had awakened him from his sleep.

A few years ago there was a tendency to operate upon everyone who had any kind of a vague grumbling distress in the right lower quadrant. The diagnosis of chronic appendicitis and the removal of the so-called "chronic appendix" was practiced extensively. Soon it became evident to the medical profession, however, that many appendices were removed which were not diseased and which were not the cause of the patient's symptoms. The pendulum tended to swing the other way. Surgeons became increasingly hesitant to remove an appendix unless they were quite certain that the patient had a very definite and acute inflammation of this organ. We fear that this tendency may have gone too far and, though we by no means recommend the routine removal of appendices in patients who have vague pains in their abdomens, we nevertheless urge immediate operation in patients who have serious pain in their right lower abdomens for which no cause other than appendicitis is evident.

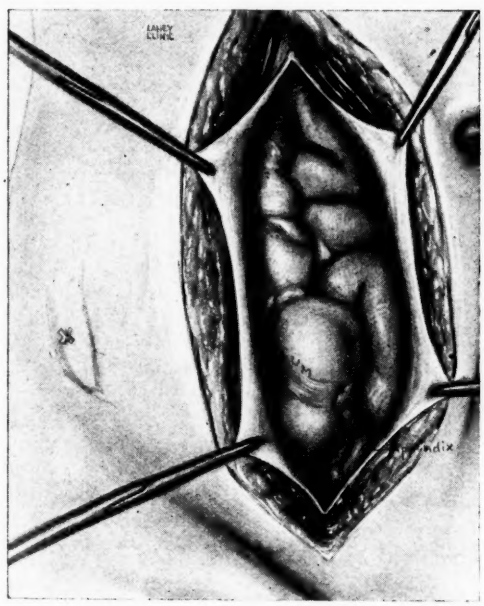

FIG. 4. In the technique of appendectomy we prefer spinal anesthesia. A long incision, four to six inches, is made in the right lower quadrant for clear vision of the area involved.

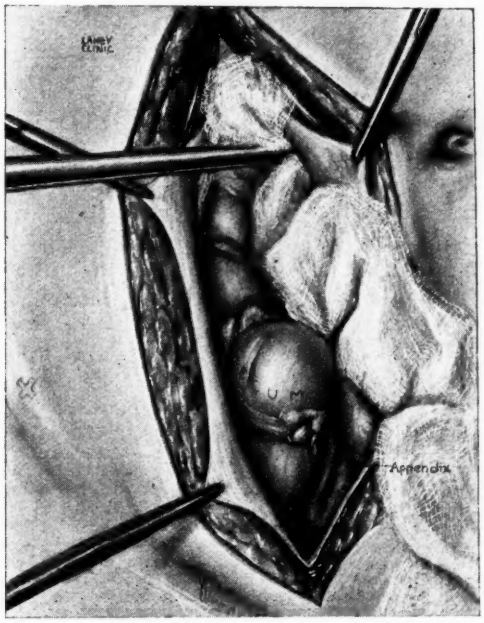

FIG. 5. After the appendix region has been inspected a wet gauze pad is gently inserted to push the omentum and the small intestine away from the region of the cecum, thus bringing the appendix into view.

Every surgeon has developed, as his experience has grown, a technique for the actual removal of the inflamed appendix. There are certain details in the surgical technique of appendectomy which, in our experience, have proved valuable and which we believe have been material factors in the reduction of our mortality in dealing with this disease. None of these details are in any way original yet we wish to repeat them at this time since we feel so strongly that they have a very definite bearing upon the results which are obtained in treating acute appendicitis.

Spinal anesthesia has been a great addition to the treatment of all inflammatory conditions within the abdomen and particularly to the treatment of acute appendicitis. With the patient under spinal anesthesia the motion of the abdominal muscles is reduced to a minimum. The intestine is markedly contracted and when the abdomen is opened lies quietly before one as though it were in the dissecting room. There is no straining, no coughing and no vomiting to push out intestinal contents and thereby tend to spread infection from the appendiceal fossa into the general peritoneal cavity. We consider that spinal anesthesia has been of life-saving importance in our treatment of acute appendicitis.

Under spinal anesthesia the abdomen is open with a large right rectus muscle retracting incision. We feel that it is most important that the incision shall be from four to five inches, or even more, in length. Nothing is more important than adequate exposure of the infected area of the peritoneal cavity so that extensive manipulation inside the abdomen will not be required. With the large incision completed the peritoneum is elevated and the interior of the abdomen, particularly the right lower quadrant, is inspected. Usually the appendix can be seen and the position in which it lies in relation to the cecum and the small gut carefully noted. A gauze pack is now gently inserted into the abdomen to push the omentum away from the right lower quadrant and to push the small intestines away from the immediate area of the lower cecum. When this has been done the cecum rolls inward and the appendix usually appears directly in vision. With a hemostat the mesentery can now be very gently grasped and the appendix can be carefully delivered into the wound. When it is in view, the wound is thoroughly draped with wet pads to protect the wall and the appendix is removed by first making a mass ligature of its mesentery. This is divided and then two ties are placed around the base of the appendix which is removed beyond them. We have not inverted the appendix stump with purse string sutures, for at least eight years and we feel that not only is this step not necessary but that it is not advisable. We believe that a purse string suture may carry infection by penetration of the thin cecal wall with the needle and that it is a piece of technique which adds to the time of the operation without giving any beneficial result. A double tie is placed about the appendix and after it has been removed and the base cauterized with carbolic acid and alcohol, the stump of mesentery is tied into the second ligature around the base of the appendix. When the stump of the appendix has been covered by the mesentery, the gauze draped about the wound is removed from inside the abdomen and the intestine permitted to fall back quietly into the right lower quadrant. The omentum now comes down and is laid in the region from which the appendix was removed. We close the abdominal wound by starting at the upper angle and putting a continuous suture in the peritoneum from above downward. In this way, as a piece of gauze is left lying beneath the peritoneum and slowly pulled out as the stitch progresses the omentum beneath this gauze tends to be constantly drawn further downward into the right lower quadrant. It is particularly important in this technique that once the appendix has been visualized no manipulation in the general peritoneal cavity is carried out at any time throughout the operative procedure.

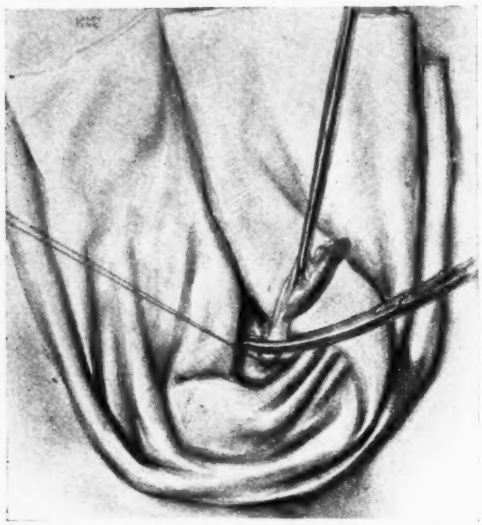

FIG. 6. The appendix is lifted into vision through the large abdominal wall incision; the field is carefully protected with wet gauze; the appendix is removed after its base has been tied with two ties. No purse string or inverting sutures are used. The stump is cauterized with carbolic acid and alcohol and the mesenteric border tied over its end.

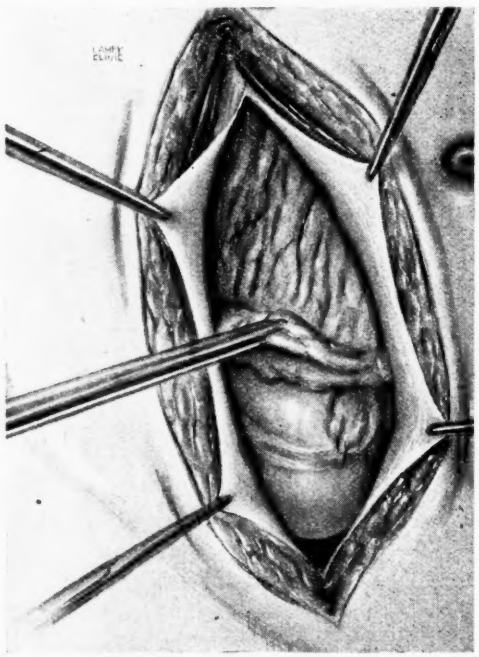

FIG. 7. The intra-abdominal gauze pack has been carefully removed and the omentum is laid down in the right lower quadrant in the region of the appendix. The wound is now ready for closure from above downward.

The third factor which we believe has a particular value in reducing the mortality of appendicitis is the management of postoperative complications. The most frequent complications after appendectomy are peritonitis, intestinal obstruction and the development of residual abscesses. It goes without saying that the early detection and the careful management of these complications will save many patients who might otherwise fail to recover.

Persistent fever after the removal of an acute-

ly inflamed appendix is commonly due to either a collection of pus in the pouch of Douglas, a collection of pus underneath the diaphragm and above the liver in the subphrenic area, or to the presence of infected material in the abdominal wound. In any case it has been our experience that persistent fever after appendectomy means constant watchfulness on our part for the presence of pus usually in one of these three locations. When it has been discovered by rectal examination, by x-ray of the diaphragm or by repeated probing of the wound, incision and drainage are indicated.

Cases of appendicitis that are rapidly fatal, however, are most commonly related to the presence of either obstruction or peritonitis or both coming on relatively soon after the operation and it is these complications that I wish to discuss at this time.

In every case in which we fear that a localized peritonitis is present when we operated upon the appendix, or in which we know that a diffuse or generalized peritonitis is present we adopt the methods of treatment known as the Murphy-Ochsner treatment. I believe it is important to call your attention once again to the details of this method since it is so advantageous to carry it out in all its details. The patient is placed sitting high in bed; he is given absolutely nothing by mouth since the intake of fluids and food by mouth stimulates intestinal peristal-

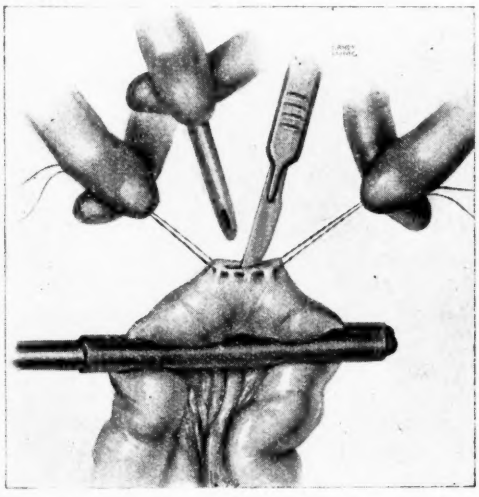

FIG. 8. Jejunostomy may be done through a two-inch incision under local anesthesia in the left upper quadrant. An intestinal clamp, such as pictured, may or may not be used. A purse string suture is placed in the intestine and a catheter quickly inserted. The purse string suture is now tied about the catheter and the suture is then drawn directly through the catheter and tied to prevent its pulling out.

sis. In the management of peritonitis it is essential that all peristalsis shall be as quiet as possible in order that the inflammatory area in the peritoneum may be walled off and resolution may take place. Morphine is given to these patients in large amounts, so that they are constantly well asleep with this drug. This not only eases the patient's discomfort but also quiets intestinal peristalsis. Furthermore, if the patient vomits at all, a Levine tube is passed at once to empty the stomach of its contents and is either left in situ or passed again at intervals of ten to fifteen hours. Large amounts of fluid are given subpectorally or intravenously. At the present time we find that

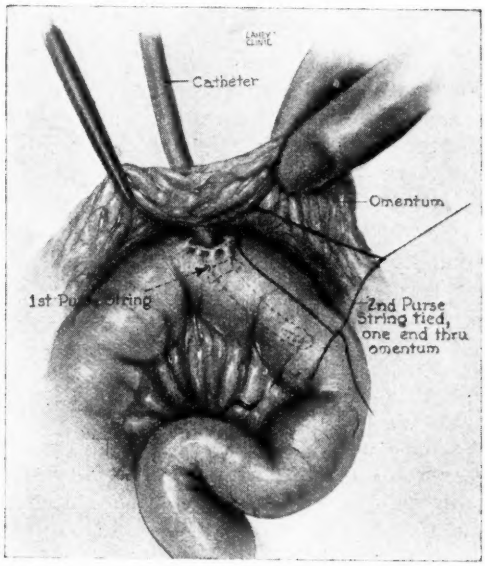

FIG. 9. A second purse string suture is placed above the first and tied. The catheter is now led through a piece of convenient omentum which is tied down to the gut with the remainder of the second purse string suture. The interposition of omentum seals off the hole in the intestine very rapidly when the catheter is removed.

constant venoclysis is the handiest and most satisfactory method of giving this fluid.

If in spite of this Murphy-Ochsner treatment distention continues and little or no active peristalsis can be heard with a stethoscope on the abdomen, we may well fear that either a spreading peritonitis is present or that an intestinal obstruction has taken place. If adequate relief of nausea and vomiting and distention does not occur from enemata; from repeated passages of the stomach tube, or from the indwelling Levine duodenal tube, we believe that jejunostomy in the left upper quadrant should be undertaken.

It is not possible to accurately analyze the results of enterostomy in peritonitis and in the obstruction which accompanies peritonitis, since each case varies so markedly in its details. It is our distinct feeling, however, that in certain cases recovery has been entirely dependent upon the fact that an enterostomy was made in the jejunum which relieved the obstruction. I, personally, am inclined to believe that in the seemingly hopeless case of appendicitis with peritonitis, an enterostomy performed under local anesthesia will, occasionally, produce unexpectedly satisfactory results.

It is our feeling that the majority of the fatal cases of appendicitis arise as a result of obstruction to the lumen of the appendix by a

fecolith or band. These patients have typical clinical histories which can be readily recognized by physicians who are familiar with the pathology of this condition. These are the cases that demand early operation in order that rupture of the appendix shall not occur and that peritonitis shall be avoided. These are the cases that result fatally when they are not recognized in their early stages.

We believe that the mortality in appendicitis can be materially reduced by the use of spinal anesthesia properly administered and the adoption at operation of a large incision permitting easy vision of the appendix area and the removal of the appendix without soiling the adjacent viscera in the abdomen.

Finally it is our belief that apparently hopeless cases of appendicitis having peritonitis and obstruction will occasionally be saved by an enterostomy.

Discussion

Chairman Miller: Dr. Clute, we thank you for this most interesting and valuable contribution on a subject which is of extreme importance.

The discussion will now be opened by Dr. Peer Johnson, surgeon, Beverly Hospital, Beverly, Massachusetts.

Dr. Peer P. Johnson, Beverly: *Mr. Chairman*—So much has been said already on the subject of appendicitis and it has been so illuminatingly covered that there seems to be little left but to agree or disagree with the reader of the last paper.

Dr. Clute has done us a very good service, I think, in calling to our attention this very insidious and rapidly fulminating type of obstructive appendicitis. It is hard for the ordinary man, the uninitiated practitioner at least, to appreciate the serious lesion which is going on with the paucity of the physical findings. As Dr. Clute said, the temperature is normal or apt to be nearly normal, the pulse rate is rarely elevated, the tenderness is slight, and spasm may or may not be present. The picture is much complicated if one happens to see these cases at a time when gangrene has taken place without perforation into the peritoneal cavity. Then the patient is comfortable, the tenderness is practically gone, there is no pain, and, in the absence of these symptoms, unless one has taken a very careful history, one is apt to be led much astray.

One point which I think is important in helping to determine the seriousness of the lesion in such cases, as in other types of appendicitis, is a careful study of the blood picture. Nothing has been said about the polymorphonuclear percentage. The white count is generally raised but not invariably. However, within a very short period of time after the onset, the percentage of polymorphonuclears may rise sometimes well above 90 per cent. I have seen it over 80 per cent within three hours after the onset of the attack. Personally, I employ this method in every case, not for the purpose of making a diagnosis, but for the purpose of determining the severity of the lesion. With the history consistent with acute appendicitis and a rising polymorphonuclear count, operation should not be delayed.

Cases which have been previously operated upon, but in which the appendix could not be removed, are also apt to prove misleading and produce serious results. Due to obstruction or impairment of circulation, the appendix is apt to perforate early in subsequent attacks. Here the physician is at a disadvantage because he sees the scar which suggests that the appendix has been removed, and the patient himself often does not know whether it has, and so the early symptoms are assumed to be due to intestinal adhesions or the beginning of intestinal obstruction. Over 10 per cent of our fatalities occurred in this type of case.

One case which recovered and which I saw within a few minutes after onset, suggested an acute intestinal obstruction. There was very little tenderness or spasm and the pain was intermittent in character. Two hours later, the abdomen was rigid, retracted, and the white count high. The abdomen was opened immediately and found to contain a large amount of free pus, with the appendix perforated. Patients, in the presence of appendiceal abscess or general peritonitis who have drainage operation only, without the removal of appendix, should always have the appendix removed as soon as conditions are favorable. If this is not feasible, they should at least be told that they still retain their appendix and the danger of succeeding attacks.

Dr. Clute's excellent mortality statistics are due, I believe, more to his efficient surgery than to any one of the technical factors which he has mentioned. He considers spinal anesthesia one important factor. To be sure nothing gives such good relaxation or so comfortable an operating field as spinal anesthesia. I feel, however, and perhaps without foundation, that it is too serious an anesthetic for what should be, in the majority of cases, a simple operation. In our own work, therefore, we have rarely used spinal anesthesia for the removal of the appendix but have depended principally upon ether and occasionally local anesthesia. In our last 529 cases in which the appendix was removed, ether was the principal anesthetic used and our mortality was 1.9 per cent. I merely quote that as a comparison with Dr. Clute's statistics in spinal anesthesia. We do not think one should use spinal anesthesia for small operations. We reserve it for operations where we have a more serious duty to perform.

As to the incision, I still prefer the McBurney incision as I feel it is farther toward the edge of the abdomen with less exposure of the small intestines. I rarely use gauze to wall off, and I still continue to bury the stump of the appendix with an enfolding stitch. The added time is negligible and the danger of cecal abscess seems to be extremely slight. It may not be of any great importance whether the stump is buried, but it gives me a feeling of greater security to have it out of the way.

I cannot too emphatically endorse what Dr. Clute said about the Murphy-Ochsner treatment. I heard Dr. Ochsner over thirty years ago give his discussion of the treatment in Boston. Since that time I have always used it in cases where there was peritoneal infection, whether it was due to the appendix or any other cause. I believe it is a life-saving measure and that many apparently hopeless cases will recover with this treatment.

During the course of this treatment, attempt to empty the bowel by enemata should be absolutely avoided. Where all is going well, enough gas is passed by the tube to keep the patient comfortable. Following the use of enemata, two cases under my observation which appeared to be doing well, suddenly showed spread of peritoneal infection. I, personally, never allow the use of enemata in conjunction with Ochsner treatment, because the danger of suddenly relieving intra-abdominal pressure with the result that light adhesions may be suddenly torn apart allowing the escape of pus from a localizing abscess.

As to enterostomy, it is undoubtedly a life-saving measure in postoperative obstruction and probably, when done under local anesthesia, does no harm in cases of paresis due to general peritonitis. However, I have never been able to convince myself that it is of the least value, and I have felt that I have accomplished everything with the indwelling Levene tube that could be done with an enterostomy.

CHAIRMAN MILLER: Further remarks on this subject will be carried on by Dr. Thomas Donovan, of the Burbank Hospital, Fitchburg.

DR. THOMAS R. DONOVAN, Fitchburg: *Mr. Chairman*—There seems very little left to be said but I do want to mention some of the factors which I believe have a bearing on this subject which may present themselves during the preoperative, operative and postoperative stages. I have noted them down, and I will read them as rapidly as I can.

I wish to thank Dr. Clute for his most interesting and instructive paper.

Fundamentally I think we can say that in these cases our objective is to prevent diffuse peritonitis and if it is present to treat it efficiently and promptly. With this thought in mind I wish to mention briefly some of the factors which I believe have a bearing on this aspect of the subject as they may present themselves during the preoperative, the operative and the postoperative stages.

In Donhauser's work entitled, "A Surgical Diagnosis," there are listed under the heading of Differential Diagnosis in Acute Appendicitis 23 separate diseases. When we consider how obscure or misleading the onset symptoms of acute appendicitis may be, especially in childhood, in old age and in women between the ages of 40 and 55, the length of this list serves but to explain in part the difficulties of making a diagnosis in many of these cases. No two individuals suffering from acute appendicitis will have all of the same symptoms or exhibit the same symptoms in the same way.

In two cases of acute obstructive appendicitis seen by me where the appendix in each case lay deep in the pelvis practically the only symptom present was an acute, persistent, bilateral pain located in the groins. One of these cases a boy, aged 14, had slight vomiting associated with his pain which was not severe. The other case a man, aged 28, had severe pain with no other symptom. In neither of these cases was there pain, tenderness or spasm in the right lower quadrant, or pain in any other part of the abdomen. Not even in cases which have gone on to abscess formation is the diagnosis always as clear as it might be.

Where the surgeon after a careful physical and history examination is left harassed by an honest doubt, unless definitely contraindicated, operation is warranted. Indecision is the ally of failure and failure spells peritonitis. This is especially true in acute obstructive appendicitis to which Dr. Clute has referred in his paper. Symptoms which call for surgical intervention at nine a. m. should not be subjected to palliative measures because it is two a. m. The use of enemas or cathartics still needs to be condemned. Rectal examination should be routined in every doubtful case. I agree heartily with Dr. Clute when he says, that in proper hands spinal anesthesia is ideal in these cases and not infrequently its use is a vital factor in successful treatment.

It is my belief that as time goes on we will see the purse-string suture gradually disappear as a part of the surgical technique of an appendectomy. Fowler's position is used for the purpose of obtaining drainage of the peritoneal cavity. A patient slumped down to the extent that the peritoneal cavity is parallel to the bed is not being helped in any way. Ileostomy should be done without delay when indicated, and in those fortunately rare but very serious peritonitis cases wherein on the fifth or sixth postoperative day out of a clear convalescent sky a persistent profuse diarrhea associated with distention, high fever and de-

lirium develop, ileostomy and blood transfusions may save the day.

Postoperative vigilance which will permit of the ready institution of proved remedial procedures or agents in the event of the development of postoperative complications, in my opinion, is just as important a factor in the reduction of the mortality rate in acute appendicitis cases as is the sum total of our best efforts applied during the diagnostic and operative stages of the disease.

CHAIRMAN MILLER: I am going to ask Dr. Frank Lahey, of Boston, if he will continue this discussion.

DR. FRANK H. LAHEY, Boston: *Mr. Chairman*—I feel sure that we are suffering today in the recent mortality rate which is prevalent over the country in appendicitis from the effects of our conscience. Our conscience, up to recently, used to bother us, I believe, because we took out so many appendices that probably did not need to come out and now, as a result of this, we tend, I think, to lean over backward. I feel sure that I do in refusing operation, even in the cases of chronic appendicitis, many of which actually do need to come out.

We were undoubtedly mistaken in the past, I believe, when we assumed that those appendices which were surrounded by adhesions represented chronic appendicitis. Dr. Clute has demonstrated definitely and certainly we have felt quite sure that the most potential appendicitis is not that with adhesions but that with a fecolith. The appendix which contains a fecolith is certainly the most dangerous type in which acute appendicitis may arise at any time, and when it does it is the type in which perforation is most likely to occur at the point where the fecolith is located, as is shown so beautifully in Dr. Clute's illustration.

There are some clinical features about appendicitis which are of value, and these are, those patients who have diffuse abdominal pain, arising in the left lower quadrant, then spreading to the epigastrium and finally localizing in the right lower quadrant are the ones in which one should be most suspicious of appendicitis. The type of pain which arises on the opposite side of the abdomen, or in the epigastrium, and then localizes in the appendix, I believe is the most definite type of pain associated with appendicitis. This is, of course, particularly valuable when it is associated with localized tenderness.

One should not place too much dependence upon the white count. I feel quite certain that the white count in appendicitis is related, not so much to an inflammatory lesion as to whether the lumen of the appendix is occluded or the appendix, itself, is becoming gangrenous. One can have quite definite appendicitis of the mural type in which the walls of the appendix are stiff and indurated but in which there is not very much toxic absorption, little or no temperature and little or nothing in the way of reactions as to white count.

There are some technical points, briefly, which I think everyone who has dealt with appendicitis cases in any considerable number has learned. On the other hand, it might be well, as a result of our experience, to state them.

I believe that all appendices should be removed under direct vision and never by palpation. When you search for appendices blindly you at times undoubtedly pull off an appendix from the parietal peritoneum which has perforated but which has adhered to the parietal peritoneum and so occluded the opening in the appendix and prevented dissemination of the contained liquid material. If you pull off this appendix from the parietal peritoneum this liquid, infected material can then run out of the appendix, be disseminated over the peritoneum, cause a peritonitis and a fatality, which might have been avoided had the appendix been visualized, walled off and any escape of pus carefully removed by a suction apparatus.

I believe that one can exhibit as much surgical judgment and technical skill in an operation for acute appendicitis as in any surgical procedure with which I am familiar. Often the margin as to whether patients live or die in an operation for acute appendicitis is a very narrow one and dependent often upon the technic of the surgeon.

Another valuable technical point is that you should not put drains on the inner side of the cecum, if you can avoid it, because if you put them there you place them in the coils of the small intestines which tend to become adherent and to produce intestinal obstruction. The ideal point at which to drain is between the cecum and the external parietal peritoneum.

I would like to add, also, a word as to spinal anesthesia because we have advocated spinal anesthesia so much and because we have made so much use of it. I would like to add again the remarks which I have always made in connection with spinal anesthesia. It is a fine anesthetic, particularly among patients with acute appendicitis and intestinal obstruction if it is given in properly selected cases and by one who is expert, not only in its administration but in meeting the emergencies which occasionally arise with it. It tends, however, to make one feel that it is safer than it really is. It is a bad anesthetic for the very ill, for the very young, for the very aged and for the patient who is a bad surgical risk. It is an excellent anesthetic for good surgical risks in the hands of anesthetists who are skilled and experienced in its use.

Finally, I believe that any patient who has demonstrated in his appendix the presence of a fecolith has plenty of evidence, at least in my

opinion, for one to advise appendectomy. I feel sure that if I had a fecolith in my appendix I would want to have it out because I believe that such an appendix is more liable to acute appendicitis than any other type, and if acute appendicitis does develop in an appendix of this type serious results not infrequently ensue.

CHAIRMAN MILLER: Gentlemen, I am obliged to request that there be no further discussion of this paper at this time. I think we will have plenty of time at the end, and if anyone wishes to make further remarks, I will ask that they be delayed until the end of the next paper. I will now request Dr. Clute to close the discussion.

DR. CLUTE: I think perhaps the best lesson that I have learned in the management of appendicitis was many years ago when my chief, Dr. Lahey, sent me out into the country to see a man who had a pain in the abdomen. I saw the man, examined him, and came back, and Dr. Lahey asked, "Are you going to operate on him?" I said, "No." He said, "He hasn't appendicitis?" I said, "No." "Well," he said, "what has he?" Since I did not know of any other cause for his pain, I could not answer him, and we operated upon him, and it was acute appendicitis. With patients having attacks of pain in the abdomen for which you can give no other explanation, one should assume that the burden of proof is on your shoulders to show that it is not appendicitis.

In closing, I want to emphasize once again that if you will cut open the appendices which you remove and examine their lumens inside you will be amazed at the frequency with which you find obstructive lesions; and, if you recall the pathology which the pictures have shown and recall the symptoms in cases as you know them, I think you will agree with us and with Professor Wilkie, who emphasized the condition so frequently, that obstructive appendicitis is a dangerous type; that its symptoms are characteristic; that an operation performed early will save the patients; and that if one delays operation serious, if not fatal, peritonitis will almost certainly result.

CHAIRMAN MILLER: Now, gentlemen, I have great pleasure in introducing our next speaker. In arranging for this meeting this morning we discussed whom we might get from outside of the State, a person of wisdom, experience, and judgment, who could best talk to us on this very important matter, and we decided to ask a gentleman, who we are delighted to say accepted and has made a long and tiresome journey from another state to be here this morning, and as the last speaker I am proud to present to you Dr. George P. Muller, of Philadelphia, Professor of Clinical Surgery in the University of Pennsylvania School of Medicine.

MORTALITY OF ACUTE APPENDICITIS*

BY GEORGE P. MULLER, M.D.†

ACUTE appendicitis is the most important of the surgical diseases which come under the observation of the physician, even more so than cancer or heart disease, because its treatment is more easily controlled under proper management on the part of the patient or physician. Perhaps 20,000 persons die each year in the United States from this disease and while some will die despite the fact that they are properly diagnosed and skilfully operated on, the majority of deaths result from delay. In a study of the appendicitis record for 1930, Hoffman (*Spectator* 127:9, 1931) tabulates an array of statistics showing the increase in the number of deaths from this disease in the United States. Objections have been raised to these figures by various writers who point to the steady decrease in the mortality rate of operation as shown by reported series but some reported series bear out the contention of Hoffman. For instance, Duggan (*Boston M. & S. J.* 192:247, 1925) showed that in the Boston City Hospital, 33 years ago, 146 patients were operated on with a mortality of 10.8 per cent whereas in 1922, 913 operations were done with a mortality of only 3.1 per cent but a computation will show that actually more deaths have occurred. There is no doubt that an increasing population and an increasing incidence of the disease are the factors.

In answer to the statement that the diagnosis is more accurately stated now than formerly the Metropolitan Life Insurance Company explains that beginning with 1911, great care has been exercised in securing complete statements of the causes of death. Peritonitis and other terminal conditions in appendicitis were not accepted without qualifying and additional information on the circumstances leading to the condition. Hence, these Metropolitan Statistics are as nearly complete as any series of data can be. In view of the fact that surgical technic has been improved since 1911, the Metropolitan Company believes that factors have arisen which increase the incidence of appendicitis, and hence raise the number of deaths. One of the things mentioned in their *Bulletin* as being of possible

*Read before the Surgical Section of the Massachusetts Medical Society at its Annual Meeting, June 10, 1932.

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importance is that of the increasing use of mineral oils.

As I see it, the problem has several angles, the most important of which are early diagnosis and prompt operation. The practitioner must learn to diagnose appendicitis in the early stage, must bear in mind that the sudden onset of abdominal pain followed by local tenderness and a rise in white cell count are the significant factors. He should know that the icebag has no influence upon the infection, that purgatives aggravate the disease and aid in the dissemination of peritonitis in the fulminating cases. We must abandon the idea that a patient operated upon within the first 24 hours is an early case; within 12 hours is a better rule and even in this time perforation with fulminating peritonitis can occur. It is true that abdominal pain may occur from food irritation or other causes which do not require surgical treatment, but this is only a matter of detail in diagnosis and does not affect the main issue. A second factor in the high mortality can be considered briefly in the statement that there is too much surgery done by inexperienced men. It is a well-known fact that the mortality of appendicitis in the large clinics is very much smaller than in the private hospitals. Lastly, I believe that much of the onus for delay rests on the public who, habituated to catharties for generations, dose themselves with oil and salts and, unwilling to submit to operation, cajole their medical advisers into procrastination. Many patients insist on having the appendicitis "frozen out" by icebag treatment. A campaign of instruction of the public regarding appendicitis is more important than the campaign against cancer. This should include the dangers of delay, and particularly the economic factor should be stressed because a drained case is infinitely more expensive to the patient and to the hospital than is a simple closed wound. In my series 35 per cent of the patients required drainage.

I believe that the recent outcry in the profession against the removal of the so-called chronic appendix is a step backward. An affected appendix should be removed always although the physician must distinguish between this and other causes of pain in the right side.

These remarks may sound like platitudes but the situation is serious in that certain statisticians are inclined to blame the medical profession for the increase in death-rate. We have succeeded in reducing the mortality in the operated cases, we can diagnose the disease, we must now put the issue up to the public. After the patient is sent to the hospital, early or late, the treatment is entirely a surgical procedure and the rest of this paper will be concerned with my own practice.

DEATH RATES PER 100,000 FROM APPENDICITIS. METROPOLITAN LIFE INSURANCE COMPANY. WHITE LIVES. INDUSTRIAL DEPARTMENT, 1911 TO 1929

Age Period	White Lives				Percentage Change 1925-1929 Since 1911-1915		Per Cent Male of Female Rate	
	Males		Females					
	1925 to 1929	1911 to 1915	1925 to 1929	1911 to 1915	Males	Females	1925 to 1929	1911 to 1915
All Ages— One and Over	15.6	13.0	11.7	10.3	+ 20.0	+13.6	133	126
1 to 4	11.5	5.1	9.2	4.8	+125.5	+91.7	125	106
5 to 9	12.3	10.9	11.9	10.1	+ 12.8	+17.8	103	108
10 to 14	14.9	15.3	10.8	12.6	— 2.6	—14.3	138	121
15 to 19	17.3	17.8	11.2	12.5	— 2.8	—10.4	155	142
20 to 24	16.3	14.9	9.6	10.2	+ 9.4	— 5.9	170	146
25 to 34	15.1	13.7	9.6	10.7	+ 10.2	—10.3	157	128
35 to 44	17.2	12.6	12.1	8.7	+ 36.5	+39.1	142	145
45 to 54	20.4	11.7	15.2	11.3	+74.4	+34.5	134	104
55 to 64	21.0	13.0	17.0	9.6	+61.5	+77.1	124	135
65 to 74	22.3	13.9	16.7	10.2	+60.4	+63.7	134	136

When we study a series of cases we invariably find that the mortality of operation for acute appendicitis is dependent upon the age of the patient, the duration of the disease, the influence of laxatives and the surgical technic. In addition there is a definite influence exerted by the virulence of the infection and resistance of the patient. For the purpose of this paper I am reporting the experience of my Clinic in the University Hospital for six years (1926-1931 inclusive).

UNIVERSITY HOSPITAL—DIVISION B.

	Cases	Deaths	Mortality
1. Chronic Cases	261	1	0.38%
Not Drained			
2. Acute Uncomplicated	361	1	0.28%
3. Acute Gangrenous	18	0	
Drained			
4. Acute Perforated or Gangrenous	35	0	
5. Abscess Localized	46	0	
6. Diffuse Peritonitis	124	11	9.0%
7. Diffuse (not operated)	1	1	
Grand total	846	14	1.7%

The operations comprised in this series were performed by me and the assistant surgeons

(Fellows in Surgery) associated with me and hence the technic was uniform throughout, no variations in the system being allowed. Young surgeons properly taught soon develop facility in technic and their total mortality has been less than that of the group operated on by me. But I believe that the occasional operator misled by the ease of removal of the simple type is unable properly to cope with the complicated case.

It will be noted that there were 585 cases of acute appendicitis with 13 deaths, a mortality of 2.2 per cent. In those under ten years of age the mortality was 3.3 per cent (61 cases) and in those over 50 years of age it was 6.9 per cent (29 cases). It did not seem necessary to study the statistics of duration of the disease because this factor is so well known, but I will quote the table in Bower's (*Bull. Phila. Dept. Public Health,* Nov., 1931) study of appendicitis in Philadelphia from reports from 28 hospitals.

eral practitioners upon the diagnosis of the disease and while the percentage of cases admitted within 24 hours was unchanged it is expected that further effort will produce appreciable results. All other considerations pale before the important one of early diagnosis and surgical treatment and while it is well to emphasize the matter of hours in the gross, we must constantly keep in mind that many patients have fulminating appendicitis in which cases even minutes are of importance. One of our Staff was operated upon six hours after onset. He took no laxative, no food and yet had 19,000 leucocytes, a phlegmonous appendix and early peritonitis.

The matter of classification seems to vary in the reported series so that some explanation is in order. In the chronic group I have placed 29 cases which were admitted, diagnosed as acute appendicitis, but which showed only slight changes such as congestion, had no leucocytosis or fever.

The uncomplicated acute cases represented

CHART NO. 1 (BOWER)

SURVEY OF 1928-29 AND 1930

Comparative Results

	1928-29	1930
Number of Cases	5,121	3,095
Number of Deaths	306	149
Per Cent Mortality	5.97	4.81
Time, onset of symptoms and operation hours	61.17	49.36
Recovered	56.5	48.67
Deaths	95.6	68.05

	1928-29			1930		
	Recovered	Deaths	Per Cent	Recovered	Deaths	Per Cent
Admitted within 24 hrs.	1,643	42	2.55	938	24	2.56
Admitted within 48 hrs.	1,648	104	6.31	1,143	47	4.11
Admitted within 72 hrs.	628	54	8.59	414	31	7.49
Admitted after 72 hrs.	896	106	11.83	451	47	10.42

The influence of laxatives is not easy to determine. Bower states that of those patients who gave a positive history of not having had a laxative, one out of every 80 died; of those who received one laxative, one in 14 died; of those who received more than one laxative, one in every seven died. In my own series no patient died who gave a positive history of not having had a laxative and the ratio of "yes" to "no" in peritonitis and abscess was five to one whereas in the simple acute group it was two to one.

During March of 1930, the Department of Public Health with the coöperation of the Philadelphia County Medical Society sent 300,000 warning stickers to physicians, who were requested to affix them to the monthly statement sent to patients. The Philadelphia Association of Retail Druggists assisted the campaign by having warning placards printed and displayed in drug stores. Radio talks were also given. Whether this campaign had any definite effect on the giving of laxatives is difficult to determine but it did focus the attention of the general grades of inflammation short of perforation or gangrene and many times there was free fluid present. But even with the addition of the non-perforated gangrenous group it is evident that in acute appendicitis without perforation, the pathologic changes being confined to the appendix, the mortality is exceedingly low, 0.26 per cent in this series (379 cases, 1 death). Thirty-five cases with distinct localization were drained and perhaps these belong in the following groups because as Ashhurst (*Ann. Surg.* 1927, 85:89) states, "if the perforation occurs into preformed adhesions the case is one of abscess or gangrene, if it does not occur into preformed adhesions, it produces diffuse peritonitis." The reason for draining these cases will be discussed later.

The localized abscesses were walled-off collections around the appendix in the subcecal space or were eccentrically placed as emphasized by Homans. The appendix was gangrenous or sloughed off and in ten instances was not located. One of these had a persisting sinus and returned for removal of the appendix about two

months later. A second patient was well for three years and then had a severe recurrence of acute appendicitis requiring operation. A third patient was well for about four and a half years and recently has had an attack but so far has not consented to operation. One patient has been lost sight of. All of the others report themselves as having had no trouble.

Diffuse peritonitis stands out as the predominant factor in the mortality, 92.3 per cent of the fatal cases having this lesion at the time of operation and 21.2 per cent of the acute group were considered to be in this class. The word diffuse is defined in Webster as widespread and hence is a correct term but the extent of diffuseness is not possible of description when correct technic is used. Certainly those of us who use the McBurney incision see only a very limited area of the abdominal cavity. We use the term when the appendix is perforated or gangrenous and without walling off, the abdomen apparently soaked with turbid fluid, a pelvic collection of thicker fluid, and changes in the color of the terminal ileum. However, it is evident that the necessity for drainage indicates an advanced lesion and in this series 205 cases were drained with a mortality of 5.4 per cent as contrasted with 379 acute cases without drainage and a mortality of 0.26 per cent, or 21 times greater.

Studies of cultures from the peritoneal fluid, as usually done, are unreliable for exact diagnosis and usually are unnecessary because of the great amount of work required for proper study. A good many of these cases have had bacteriological examinations but I prefer to quote from the investigation made a few years ago by Meleney (*Ann. Surg.* 93:937, 1931): "B. coli, Green Streptococci and B. welchii, were the organisms most commonly found. There seemed to be no association between the type of organism and the severity of the lesion but rather it is the dosage of these organisms and the presence of irritating intestinal juices that determine the fatal outcome."

Time for Operation. I believe that every case of acute appendicitis should be operated upon at once if the condition of the patient demands it. In certain of the milder cases we have delayed operation when the patients have been admitted after midnight and careful scrutiny of this group shows that in 414 cases only one might have escaped drainage had operation been done immediately instead of in 11 hours. In two other cases requiring drainage delay was practiced but in both the diagnosis was uncertain on admission. Of the 35 acute cases requiring drainage, operation was performed immediately in 32. The death in the group of 361 uncomplicated acute cases occurred in a child four years old operated on two hours after admission. In the localized abscess group, 36 of the 46 were operated on immediately and only six after 24 hours but here again the matter of diagnosis always was the factor in holding up operation, and as no deaths and no intestinal obstruction occurred in this group the delay was unimportant.

The most serious decision must be made in those cases recognized before operation as diffuse peritonitis. Bilateral rigidity, distention, and a rapid pulse are present and in the extreme cases the blood pressure is low and the patient shows a cyanotic tinge. Ice-cold hands with a high temperature has invariably been significant of a hopeless infection. Some surgeons advise delay when distention predominates over rigidity, but I have not found this decisive enough and prefer to evaluate the patient's general condition, especially his circulatory reaction. If recently, he has had purgatives, is distended with only tinkling sounds, has poor circulatory response and is admitted two to three days after onset we are inclined to delay. A low temperature and slight leucocytic response are also significant. We operated immediately in 78 per cent of the cases of diffuse peritonitis with a mortality of six per cent; after a short delay in 17 per cent, with a mortality of 14 per cent; and after 24 hours in five per cent with a mortality of 33 per cent. During the delay period the patients are given water, stimulation and sufficient morphine to relieve pain. Lately we have been giving salt solution and glucose (5 per cent) by continuous intravenous infusion, hypodermoclysis of salt solution being of somewhat less value. Adrenalin and caffeine are the only drugs used for stimulation of the circulation. I think that heat or cold to the abdomen are of no benefit. Nothing else is given, no water by mouth and no enema. We have had no experience with the use of B. welchii antitoxin. After improvement has definitely occurred, and time in hours is never considered, operation is undertaken. In this series death occurred only once and that was in a patient not operated on, admitted in extremis, and dying in 24 hours.

Anesthesia. As a general rule we use gas anesthesia with local infiltration. Often a certain amount of ether is used and occasionally local novocaine alone. In January, 1929, we began to use spinal anesthesia but are unable to confirm the reduction in mortality reported by some clinics, the mortality in the peritonitis group showing 8.7 for inhalation anesthesia and 16.7 for the spinal. Certainly this method of anesthesia is ideal for the surgeon but as our operation rarely requires more than 20 minutes we do not like the prolonged anesthetic period. Furthermore, it does not seem wise to risk, in a case of diffuse peritonitis, the low blood pressure often produced by spinal anesthesia.

Incision. Almost without exception we use the McBurney incision. It gives an ideal exposure and if properly managed never exposes

the coils of intestine except a few inches of the terminal ileum. In the simple type of acute appendicitis, after the peritoneum is opened, the omentum if interfering, is pushed aside with gauze tape and the cecum brought into the wound. The appendix is delivered and removed. Except in those cases which are plastered against the cecum or adherent against the fossa or in unusual situations, the procedure is quite simple. In some cases an edema of the subperitoneal fat will warn you that the abscess is directly beneath and in such cases the internal oblique muscle is spread further out (laterally) and the abscess opened in this situation. Then, according to circumstance, I merely drain or push in further after the appendix. It all depends upon whether I feel I can reach it easily. In other cases when the peritoneum is opened, the mass will be felt usually with the omentum adherent and with agglutinated small intestine. In such cases we push in some of our gauze tape to act as a walling-off medium and then with the aspirating tip in one hand commence finding a place of cleavage, somewhat after the manner of operating for pus tubes. As soon as we break into the abscess it is sucked dry. Again we determine whether to remove the appendix. The cavity is then filled with two or three Mikulicz or Penrose type of drains.

In the case of diffuse peritonitis one is greeted by a gush of turbid fluid and with the aspirator in place constantly sucking the pus, the appendix is sought for and removed because as it is always perforated or gangrenous it would act as a culture tube for further bacterial invasion. If we operate for diffuse peritonitis I believe the appendix should be removed. In only one case where the combination of abscess and peritonitis existed, we could not locate the appendix and simply put in drainage. This patient died. Two tube drains are placed into the pelvis and another one upward to the diaphragm. Occasionally a Penrose drain is used in addition largely as a plug to hold the wound open. In those cases in which the appendix or abscess is median to the cecum and beneath the ileum, great care should be taken not to injure the blood vessels of the ileum. Often we do not deliver the cecum, but ligate the meso-appendix and appendix in the depths of the wound. Sometimes the stump is ligated only and not inverted, but sufficient stump should be left to allow a cauterization of the mucosa, or a fecal fistula is liable to result. It is better to bury the stump if it can be done, and in the more acute cases with a soft, edematous cecum, the Gould infolding suture is better than the purse string suture. When the omentum is wrapped about the appendix, we often ligate and divide it, removing the appendix with the enveloping omentum, thus avoiding exposure of a perforation and sure soiling of the wound. The most difficult cases to handle are those where the perforation is close to the cecum, and everything is tight. In such cases it is necessary to grasp the tip and cut the appendix out, putting successive Kelly hemostats on the meso-appendix. When the perforation is reached, the appendix usually pulls loose, but with the aspirator working, no harm results. The stump is then trimmed and inverted with the Gould suture, without ligature. When the appendix is deep and adherent to the sigmoid, one has a real job on hand. But patience and good exposure will solve the difficulty. We never have any trouble with the high posterior appendix so often mentioned with dread by surgeons. If hard to reach, the muscles may be cut upward until there is free exposure, and then, if necessary, a tape is passed around the base of the appendix and used as a retractor. Curiously, these cases rarely bleed much, although occasionally loin drainage has been used for free bleeding from a high abscess with necrotic walls. A large sponge should never be placed in the abdomen, and we rarely use anything except one or two pieces of narrow gauze packing to keep the omentum away and the cecum rolled over a trifle. The idea that two or three large pads wall off the abdomen from bacterial contamination is a pure delusion.

The indications for drainage and the technical management of drains is perhaps the most interesting feature of the treatment of appendicitis. I think you will acknowledge that the general peritoneal cavity cannot be drained. Drainage, therefore, may be considered as having only a local interest. If the pus is thick and therefore largely composed of dead leucocytes, if it contains definite flakes of coagulated lymph, the indication for drainage exists. If the peritoneum covering bowel, fossa, or pelvis appears injured and covered with adherent patches of exudate, the process of healing will result in free discharge of pus, requiring an outlet. If the fat of the meso-appendix or the tips of the omentum are necrotic, the dead tissue will float off later. If an abscess has been entered, its walls will appear necrotic and bleeding. Such conditions will need drainage. Generally it is quite unnecessary to drain for simple turbid fluid, however, and never necessary in the presence of an acute condition of the appendix, even a gangrenous one if there is no spreading infection as evidenced by the absence of reaction.

I believe, without good experimental evidence, that drains act largely through their foreign body effect, thereby provoking reaction. At any rate they should not be disturbed until the well-known time for the discharge of exudate has arrived which is usually from five to ten days. We never remove a tube at the end of

five days as is so frequently advised. They are shortened from day to day. In the analysis of this group of cases I was disappointed to note that we had a secondary abscess develop eleven times. One of these patients was closed without drainage but returned two months later with a pelvic abscess. Four others had diffuse peritonitis and recovered, but subsequently had secondary collections; perhaps this was only what we could expect under the circumstances. Four other cases of abscess and one gangrenous acute case had to be reoperated for secondary collections and I feel that these probably resulted from improper handling of the drains. There is a great tendency on the part of the younger men to remove drainage too quickly. One patient only, developing a secondary collection, died. This patient had intestinal obstruction and then septic pneumonia.

We had eleven cases of intestinal obstruction complicating diffuse peritonitis of which seven died. In three cases enterostomy was performed on the following day for increasing distention. In the others, the obstructive symptoms did not develop for several days but the longest period was eight days. There were two of six days', one of five days' and two of four days' duration. We considered that these were directly the result of the peritonitis, and not purely mechanical by adhesion with angulation. All of these patients had an enterostomy performed and four recovered. I have never been able to convince myself that the primary enterostomy was of great importance because it only drains a few feet of intestine at the most, the remainder being tied up by the peritonitis. We have not delayed until the patients are moribund or until fecal vomiting has occurred, and believe that the best results are obtained in those with active peristalsis. An enterostomy can hardly be of benefit in a silent abdomen. Every effort should be made to keep all drainage lateral to the coils of intestine.

Pulmonary complications occurred 14 times, including bronchitis, atelectasis, pneumonia, infarct and empyema. Curiously the diffuse peritonitis group showed the lowest percentage of pulmonary complications. This is probably accounted for by the fact that they die so soon from the peritonitis. We had one case of liver abscess in the group, which was opened and drained 25 days after the initial operation and recovered. One of the cases of empyema recovered and one died. There was only one fecal fistula and this occurred in the patient who also had subdiaphragmatic abscess and empyema. Frequently, the interns have suggested the presence of a fecal fistula when the odor and drainage were merely microbic in origin.

Postoperative Management. All patients are given an abundance of water by one of the usual methods; proctoclysis being used for the milder cases; hypodermoclysis for the more severe and continuous intravenous treatment for the very sick. Morphine is given frequently and it is my impression that a toxic patient without circulatory depression is improved by the very free use of morphine and not hurt by it. In some cases I have given 1/6 of gr. every two hours for ten or twelve doses. We watch the respiratory rate and use this as the index of morphine saturation. Nothing else needs to be added to this postoperative treatment except to state that I think I am slower in starting the use of water and food by mouth than most surgeons. Purgatives are never administered to any case of appendicitis until at least a week has elapsed since the time of operation and never if drains are still *in situ*. When covered gauze drains are used they are removed about the fifth day and as rubber tubes are always used in addition, nothing else is done at this dressing. On the following day gentle irrigation is begun and the tubes slowly shortened until out. We douche the wound liberally with one of the newer antiseptics in order to prevent secondary infection with staphylococci. Enemas are used to move the bowels until all drains are out at which time liquid petrolatum is substituted.

From my experience I believe that as the teaching of early operation has been in vogue among surgeons since the time when Murphy, Ochsner, Deaver and the rest were writing their forceful papers, the medical profession must know the proper procedure by this time. Certainly they have been taught the correct treatment in the medical school and have heard it from the surgeons in the hospitals. We cannot conceive that the medical profession is deliberately derelict in this matter. Hence, it follows as a matter of course that the general public are the ones that must be reached by the campaign of education and taught considerable of the nature of the disease, and the inherent dangers of delay. Also, I believe that as we will always get a certain percentage of the advanced group, special studies should be made upon the best way of handling such patients. As the mortality from diffuse peritonitis is high even in the best clinics, it follows that we are unable to cope with the infection as successfully as we would hope with present methods. These two points, namely, the education of the public and the restudy of the best means for treating diffuse peritonitis, should be the immediate ambition of the medical profession.

Discussion

Chairman Miller: Thank you, Dr. Muller. A few weeks ago I was in New York, spending the morning at the Bellevue Hospital, and there I heard one of the young surgeons speak

about his method of handling the acute appendix, together with certain other abdominal operations, and he for many months had been giving in his cases of acute appendicitis an injection in the form of pituitrin, called pitressin, before he started the operation, and thereafter he gave this pituitrin hypodermically two, three or four times a day for several days. He said it had a splendid effect, did not stir up peristalsis, did not spread infection, and kept the intestines in a state of constant tone, and he was convinced it was an excellent adjunct to his other means of handling these cases.

I was not convinced then, and I am not convinced today, but there is a man doing the emergency surgery in one of the very largest New York Hospitals using this drug. I would not do it. I was not persuaded. I am going to ask Dr. Muller to speak about that when he closes his remarks.

The discussion of this paper, gentlemen, will be opened by Dr. Irving J. Walker, Clinical Professor of Surgery at the Harvard Medical School and Visiting Surgeon at the Boston City Hospital.

Dr. Irving J. Walker, Boston: *Mr. Chairman*—I suppose the chief question in the discussion of this paper, and the reason for dwelling upon this subject today is the question of the "rising mortality" in appendicitis. Is it rising? According to the literature of late years by reputable observers, we are led to believe that it is. If one analyzes that literature carefully, it will be found that the mortality is rising per 100,000 population approximately 20 to 30% over a fifteen-year period. To the same end, quoting Willis, and during the same period of time, the mortality in the treatment of gastroduodenal ulcer has risen 72%, that of gall-bladder disease 77%, and that of thyroid disease 250%. This is easily explained from the fact that surgery for the above-named conditions, and I believe the same applies to appendicitis, may have increased, theoretically at least, in the same proportion. Bear in mind that these mortality figures are based upon the mortality per 100,000 population, and not upon the incidence of the disease or operation. This, I believe, is the reason for our being somewhat disturbed over the so-called rise in the mortality of appendicitis.

In a search of the literature, we find it quite impossible to ascertain the exact incidence of appendicitis for any period. This being so, it is equally impossible to determine the mortality based upon this incidence. I feel that we should lay more stress upon the mortality based on the incidence of the disease than upon the mortality derived from vital statistics.

Our interest in this subject was aroused about one and a half years ago and has resulted in a study of appendicitis at the Boston City Hospital. We have stressed the subject of acute appendicitis, thinking that if the mortality were rising it would probably demonstrate itself in this type rather than in chronic appendicitis. We have taken the period from 1907 to 1910 inclusive, and compared that with the period from 1927 to 1930 inclusive, feeling that such a comparison would show whether the mortality was rising. Most of the writers who speak of the rising mortality in appendicitis end, as we are going to end, by quoting figures which prove that the mortality based upon the incidence of operation for the disease has not risen, and as a matter of fact is not alarmingly high. Our mortality in chronic appendicitis for the years 1907 to 1910 inclusive was 2.9%, and that for the years 1927 to 1930 inclusive was 1.2%. Our mortality in chronic appendicitis was therefore lower.

The question of the mortality in acute appendicitis was more interesting from a statistical standpoint. This I will briefly show in the following slides.

You will note by comparison that the incidence of operation for acute appendicitis has risen slightly less than 300% during these twenty years. The mortality during that period of time has dropped from 13.1% to 5.8%. Incidentally statistics gathered from many writers show that the present mortality in acute appendicitis is for the most part between 5% and 6%. You will note also from the above slide (Mortality By Years), that during the last four years studied there has been a gradually decreasing mortality.

This slide shows the mortality for the different types of appendicitis operated on in the two periods studied. This will show a diminishing mortality in all types of acute appendicitis for 1927 to 1930 inclusive as compared with 1907 to 1910 inclusive.

This slide is a personal interpretation, derived from a study of the records, of the factors contributing to the causes of death during the two periods. It will be noted that delay is still the greatest factor contributing to the mortality of appendicitis. During the second period studied, postoperative complications were a considerably greater factor contributing to the mortality than in the first period. Our only explanation for this is that of keener observation of these complications during the second period.

It is our conclusion that while the mortality of appendicitis based upon the number of deaths per 100,000 population has risen, the incidence of operation for appendicitis has markedly increased but the mortality based on this incidence has not increased.

Chairman Miller: I am now pleased to call on Dr. John Homans, visiting surgeon at the Peter Bent Brigham Hospital in Boston.

Dr. John Homans, Boston: *Mr. Chairman*

MORTALITY BY YEARS

	Total	Recovered	Died	Mortality
1907	157	132	25	15.9 %
1908	192	162	30	15.6 %
1909	180	159	21	11.6 %
1910	230	206	24	10.4 %
1907 - 1910	759	659	100	13.1 %
1927	526	491	35	6.6 %
1928	493	460	33	6.6 %
1929	492	466	26	5.2 %
1930	595	566	29	4.8 %
1927 - 1930	2106	1983	123	5.8 %
Both groups 1907 - 1910 1927 - 1930	2865	2642	223	7.7 %

MORTALITY FOR TYPES OF APPENDICITIS

	Acute unruptured			Acute unruptured with fluid (not peritonitis)			Appendix abscess			Acute with diffuse peritonitis			Acute with general peritonitis		
	Total	Died	Mort.	Total	Died	Mort.	Total	Died	Mort.	Total	Died	Mort.	Total	Died	Mort.
1907	41	0	0 %	13	1	7.7 %	57	9	15.8 %	35	10	28.6 %	11	5	45.4 %
1908	64	1	1.6 %	8	2	25 %	77	12	15.6 %	29	7	24.1 %	14	8	57.1 %
1909	67	3	4.5 %	11	2	18.2 %	73	8	10.9 %	22	3	13.6 %	7	5	71.4 %
1910	92	3	3.3 %	9	0	0 %	82	8	9.7 %	38	7	18.4 %	9	6	66.7 %
1907 - 1910	264	7	2.6 %	41	5	12.2 %	289	37	12.8 %	124	27	21.8 %	41	24	58.5 %
1927	313	7	2.2 %	47	1	2.1 %	85	7	8.2 %	46	4	8.7 %	35	16	45.7 %
1928	275	3	1.1 %	62	1	1.6 %	91	9	9.9 %	31	9	29 %	34	11	32.3 %
1929	297	4	1.3 %	62	0	0 %	66	9	13.6 %	52	8	15.4 %	15	5	33.3 %
1930	289	4	1.4 %	94	2	2.1 %	86	7	8.1 %	75	3	4 %	51	13	25.5 %
1927 - 1930	1174	18	1.5 %	265	4	1.5 %	328	32	9.7 %	204	24	11.8 %	135	45	33.3 %

and Gentlemen—As one might expect, the Professor of Surgery in the first medical school in this country and in the city where they have the lowest mortality for appendicitis has expressed himself with admirable good sense. I cannot imagine a paper which is more thoughtful and conclusions which are more to be respected than those which you have just heard.

FACTORS CONTRIBUTING TO MORTALITY

Delay - D
Faulty Surgical Management - FSM
Preoperative Complication - PR.C
Post Operative Complication - PO.C

	Total Deaths	D		F S M		D plus F S M		PR.C		PO.C		D plus PO.C		PR.C plus FSM		D plus PR.C		PR.C plus PO.C		D plus FSM plus PO.C	
	100		%		%		%		%		%		%		%		%		%		%
1907 - 1910		41	41	28	28	18	18	1	1	9	9	2	2	1	1						
1927 - 1930	Total Deaths 123	62	50.4	10	8.1	10	8.1	1	.8	25	20.3	11	8.9			2	1.6	1	.81	1	.81

I was interested to see that he laid stress upon the management of the case and particularly upon technic in keeping down mortality, for I think a good many of us have come to feel that we all understand the management of these cases and our technic is satisfying.

I was interested to see that he operates with the use of suction, and I feel that if we were permitted only one means of preserving both the peritoneal cavity and the wound from infection, one would use suction rather than walling off with gauze. It seems to me that the operation performed with the aid of the suction tube constantly at hand is likely to save complications.

As to the matter of drainage, I have always thought that perhaps the presence of dead tissue, sloughing tissue in the region of the cecum or in the omentum was perhaps a more important guide as to whether one should drain than the quality of the pus, but I daresay the two things go together.

I wish that in the consideration of statistics more could have been said about age. I had supposed that the mortality from appendicitis came especially in the very young and in the old. I have heard no one say anything about mortality in the very young except in a very sketchy way. I should like to add something about the mortality in those over 50. I took the trouble to look up all patients who have been operated on for acute appendicitis, without regard to whether there was perforation, at the Peter Bent Brigham Hospital for the last 15 or 20 years. I found 129 cases, and among these there was a mortality of about 15 per cent.

It seemed to me as I considered the fatal cases that it was not entirely a matter of failure of diagnosis on the part of those who sent the patients to the hospital. Indeed, I felt on the whole that those who referred the patients to the hospital had done so with reasonable promptness and had made as good a diagnosis as we who had seen the patients could make. Of those 19 fatal cases three or four were studied for several days before they were operated upon. They were studied because there were indications of other diseases. That is to say, of those cases three appeared to be cancer, three appeared to be suffering from chronic obstruction, and one other case presented the symptoms of colitis. So, of the 19 cases, if one were trying to lay the blame for delay in the treatment of three patients upon any one, it must be upon the members of the surgical staff of this hospital who treated them.

Again, there were three patients in the list whose deaths could be attributed in some degree to diabetes, and those patients all had sloughing wounds, the infection was uncontrollable, and nothing could be done.

There were nine instances of abscess. If one is to believe that death comes from diffuse peritonitis in people over 50, one must admit that here, at least, half of the patients in the series did not die of diffuse peritonitis but after having abscesses which were fairly well walled off and drained.

I think a good many of these individuals had come to the end of their day and the reason they died of appendicitis was because they had given out, particularly as to their kidneys and hearts, and their resistance in general was somewhat lowered. We should not comfort ourselves too much with that, but that is one reason, I think, for the higher mortality in the elderly patients.

I must draw the conclusion in comparing our mortality here with that of Philadelphia, of course, any surgeon from Boston now has to stand with his head hanging down as compared with the surgeons of other cities, and fortunately most of the members of the Massachusetts Medical Society who are present do not come from Boston, and they must feel much better than those who come from this city. I think we must admit that we do not know very much about the early symptoms of appendicitis. Evidently, we think we know more than we do, and that applies not only to the general practitioner but to the surgeon as well. Indeed, what we have always held to be the typical symptoms are not the commonest ones, a matter which we should do well to bear in mind.

I do not feel quite like laying all the blame upon the patient and the catharties, however foolishly the former uses the latter; but the more I see of appendicitis the more I realize that it is worthy of intense study, that it imitates almost every other abdominal disease, and I believe that a meeting of this sort does more than clear the air. I think it stimulates us all to pursue our investigations and modestly admit that we can still learn a good deal about this subject.

CHAIRMAN MILLER: Gentlemen, I am extremely sorry but we cannot allow further discussion at this time. It is 20 minutes past 12, and the Annual Discourse takes place in this room in 10 minutes. The Secretary and I wish to thank you very much indeed for the interest which you have shown in this subject and for the extremely satisfactory attendance.

Now I shall ask Dr. Muller if he will be kind enough to close the morning discussion.

DR. GEORGE P. MULLER: I included the remarks regarding the deaths per 100,000 because Hoffman is statistician for the Prudential Life Insurance Company and writes a good deal and his writings are being quoted.

I have had no experience in the use of pitressin in peritonitis or in appendicitis. Several years ago we were using it in connection with spinal anesthesia, but I have abandoned it for ephedrin or adrenalin. If I heard correctly I understand that the physician quoted has had no mortality as the result of using pitressin. How can this be so? Can pitressin absorb pus, can it absorb or dissipate microörganisms in the peritoneal cavity? It is infection that kills the great majority of patients who die from the toxemia of the spreading peritonitis, a few from intestinal obstruction, etc.

I think that enterostomy may be life-saving in those patients in whom peristalsis is active and obstruction definitely present. When the abdomen is simply swollen from the distention of bowel injured by peritonitis and inactive then one only drains a loop or two. I agree with Dr. Homans that the mortality in patients over 50 years of age is well above the average. We found this to be so in our gall-bladder group and in our lung abscess statistics.

Regarding chronic appendicitis, we can all agree that although pathologically speaking there may be no such entity as chronic appendicitis, yet the patient has adhesions from a previous acute attack and obstruction from concretions, or an appendix buried in congenital adhesions. In all of these cases symptoms will result and do constitute chronic appendicitis. Therefore, it is simply a matter of correctness in diagnosis. If the patient has a twisted mobile cecum, a ureteral kink, an intercostal neuralgia, etc., operative removal of the appendix naturally does not cure because he never had appendicitis as the basis of his symptoms. Generally, I am well pleased with the end-results of my group of patients operated upon for chronic appendicitis.

DR. HILBERT F. DAY, Cambridge: I wish to say a word in discussion of Dr. Clute's paper. One of the factors which I believe would, in a way, reduce mortality is a more careful attention paid to the cases which are sent into our general hospitals pre-diagnosed as appendicitis and which often go home unoperated, because when seen in the hospital their symptoms have subsided and the previous history, for one reason or another, discounted. All of us who are in charge of surgical services in hospitals see the acute, seriously ill case, often with a ruptured appendix, come into the hospital and upon investigating such cases find that they had previously spent several days in a hospital and had gone home reassured that the diagnosis was not appendicitis. Therefore, when similar symptoms reappear, the patient does not pay serious attention to them, does not promptly call a doctor and so arrives at the hospital late, perhaps too late.

DR. LESTER R. WHITAKER, Boston: I should like to mention, briefly, two points on technic; first, with regard to the length of the abdominal incision, and secondly, with regard to inverting the stump of the appendix.

I know that most surgeons advise the so-called adequate abdominal incision, but a short incision has advantages. Many times in these cases the wound becomes infected by colon bacillus and with a long incision the danger of hernia is great. I have seen a patient with scarlet fever where the wound sloughed and increased to twice its length, with the coils of intestine being visible—and in this case the incision was made as small as possible.

With spinal anesthesia there is such good relaxation that by the use of a focusing headlight and ribbon retractors the whole lower abdomen can be explored visually, without trauma to vis-

cera, through a two-inch incision. Many times with proper instruments the appendix can be safely removed through this incision even though it cannot be delivered. Thus, the abdominal wall is less damaged and the danger from breaking open of the wound, with evisceration and resultant peritonitis, is minimized.

The question of inverting the stump of the appendix has been much discussed. If you had an opening in the cecum you would not tie a string around it, cauterize it and drop it back into the abdomen. You would invert it with a stitch opposing peritoneal surfaces. It seems to me this should be done with the stump of the appendix if possible. One of the reasons given for not burying the stump has been the supposed danger of an abscess of the cecal wall.

At the Massachusetts Memorial Hospitals we have devised a new method of treating the stump to obviate this possibility, by the use of electrosurgery. The base of the appendix is crushed to the cecum. A tie of coarse silk or linen is placed around the appendix a half centimeter distal to the line of crushing. Thus the tissue is crushed below the tie. The appendix is cut off a half centimeter distal to the tie with the cutting current of the electrosurgical unit, proceeding slowly to cauterization. Then the whole stump is fulgurated with the coagulating current, including the crushed tissue below the tie. The devitalized stump is inverted with a double purse-string suture. As shown by animal experimentation the devitalized tissue sloughs into the cecum after 12 to 24 hours, doing away with the possibility of an abscess of the cecal wall.

CHAIRMAN MILLER: In closing, I request that you show your appreciation of Dr. Muller by a rising vote of thanks.

REPORT OF NOMINATING COMMITTEE

The Chairman during the session appointed the following Nominating Committee: Ralph W. French, of Fall River, and Irving J. Walker and George D. Cutler, of Boston.

The Committee retired and returned with the following nominations:

For Chairman, Allen G. Rice, Springfield.
For Secretary, Ernest M. Daland, Boston.

Upon motion, duly made and seconded, it was

Voted: To accept said report, and that the Secretary be instructed to cast one ballot for said nominees.

The Secretary having cast a ballot as directed, the Chairman declared Dr. Allen G. Rice and Dr. Ernest M. Daland elected to their respective offices for the ensuing year.

HIGH DEATH RATE OF APPENDICITIS

In a recent issue of the *Spectator*, Dr. Frederick L. Hoffman asks that more attention be given to the high death rate in this country from appendicitis. Citing the appendicitis mortality of sixty American cities from 1910 to 1931, inclusive, he shows that there has been practically no change in the mortality rate during the past ten years. Moreover, the rate for this aggregate population of over 28 million, namely, 17.4 per 100,000, is more than double that prevailing in England and Wales (7.2), and likewise more than double that in a group of 64 foreign cities (8.0) with an aggregate population of over 56 million.

Philadelphia, it will be noted, has the lowest rate in this group of cities. It is possible that this relatively good showing may reflect the interest which has been taken by that city in the subject of appendicitis. In the November 21, 1931, issue of the *Weekly Bulletin* we published a note on the two surveys of appendicitis in Philadelphia, one covering the clinical records of 5,121 patients in twenty-seven hospitals during the years 1928 and 1929, the other covering 3,095 patients in 1930. In the earlier group of cases the case fatality was 5.97 per cent; in the later group 4.81 per cent. If we assume a case fatality rate of 5 per cent, the 1,157 deaths charged to appendicitis in New York City in 1931 would represent over 23,000 cases, surely a large field for any program looking to a reduction of preventable mortality. — *City of New York Department of Health Weekly Bulletin.*

MORTALITY RATES

Telegraphic returns from 85 cities with a total population of thirty-seven million for the week ending August 13 indicate a mortality rate of 9.4 as against a rate of 9.8 for the corresponding week of last year. The highest rate (16.2) appears for Wilmington, Del., and the lowest (4.7) for Fort Wayne, Ind. The highest infant mortality rate (16.4) appears for Nashville, Tenn., and the lowest for Cambridge, Mass., Des Moines, Iowa, Duluth, Minn., Evansville, Ind., Fort Wayne, Ind., Long Beach, Calif., Lynn, Mass., Peoria, Ill., Salt Lake City, Utah, Somerville, Mass., Spokane, Wash., and Waterbury, Conn., which reported no infant mortality.

The annual rate for 85 cities is 11.5 for the thirty-two weeks of 1932, as against a rate of 12.4 for the corresponding period of the previous year.

A NEW PROCEDURE FOR OBSTETRICAL ANALGESIA

BY HAROLD H. ROSENFIELD, M.D.,* AND REUBEN B. DAVIDOFF, M.D.*

THE purpose of this paper is to submit a preliminary report upon a new procedure for inducing obstetrical analgesia and amnesia. This method is based upon the use of the newer barbiturates supplemented by the rectal administration of paraldehyde. The advantages of this procedure are as follows:

I. Production of a prolonged amnesia and analgesia.
II. Freedom of danger to the mother or the fetus.
III. Reduction of excitation to a minimum.
IV. Avoidance of delay in labor.
V. Simplicity of administration.

Until recently the two methods most widely used to obtain obstetrical analgesia have been the morphine and scopolamine régime and the Gwathmey synergistic rectal analgesia[1]. In spite of the fact that both these procedures have had ardent advocates, they have never been generally adopted because of various well-known disadvantages.

The recent literature contains many articles concerning the use of sodium isoamylethylbarbiturate (sodium amytal) and pentobarbital-sodium (nembutal) alone and in various combinations with morphia-pantopon and scopolamine. Although in most instances satisfactory amnesia has been obtained, there were sufficient drawbacks to each combination to cause some dissatisfaction. Sodium amytal or nembutal in large doses produces a rapid and prolonged sleep throughout labor; but each is accompanied by various degrees of excitation on the part of the patient, which militated against the entire desirability of these drugs, even though the infants remain unaffected, labor is not prolonged, and the amnesia is excellent. If morphia or pantopon is used to control the excitation, the infants are slow in breathing. Again, if either the combination of sodium amytal and scopolamine or that of nembutal and scopolamine is used, each produces excellent amnesia, but in most instances causes marked excitability, necessitating considerable effort on the part of the attendants to prevent the patients from falling out of bed or otherwise causing injury to themselves. The physical exertion which these patients undergo must be considered as an added burden and a contraindication, particularly in cases of cardiac disease. Another undesirable phase of the excitement caused by the above medication is the tendency of the patients to resist violently when it becomes necessary to supplement the analgesia by ether or gas-oxygen inhalations. If morphia or pantopon is used in the scopolamine combination, patients are much more quiet, but here again the infants show the effects of these drugs by definite respiratory depression. It has been attempted by some to overcome this excitement phase by the administration of rectal ether, but with results that in our experience were not satisfactory. On the other hand, a combination of the barbiturates with a drug which would augment their action, reduce the undesirable excitement to a minimum, without endangering the mother or the fetus, or delaying labor, would seemingly be ideal. A trial series of fifty cases demonstrated that paraldehyde fulfills these requirements. While we realize that this is too small a number from which to draw final conclusions, the results were promising enough to warrant this preliminary report.

According to Cushny[2], paraldehyde is a polymer of ethyl-aldehyde, resembles alcohol in its effects, though it is a much more powerful narcotic, and rarely induces any symptoms of excitement. It does not affect the heart directly even in large doses and has no such effects on the protein metabolism as have been observed under the prolonged administration of chloral. The pulse is slightly slower and the carbonic acid exhaled is less than normal. But these changes are due to the muscular movements being lessened and are hardly greater in extent than the changes that occur in natural sleep. Very large quantities have been taken without fatal results and in fact without any more serious consequences than prolonged unconsciousness. It is excreted in part by the lungs and mainly in the urine.

Our procedure has been as follows:

If the patient upon admission to the hospital is in active labor, following the routine preparation and enema, she is given seven and a half grains of nembutal. This dosage produces sleep in from ten to fifteen minutes. If contractions are weak but troublesome, we substitute six grains of sodium amytal, because nembutal may stop labor if it is not well established, whereas the sodium amytal has no such effect.

If nembutal is used, it is followed in from fifteen minutes to one half hour by rectal instillation of four to six drams of paraldehyde in one and one half ounces of olive oil. We base the amount of paraldehyde roughly upon the weight of the patient. We have found that this rectal administration is simplified by the use of the apparatus designed and described by McCormick[3]. By means of this apparatus the mixture can be injected very rapidly and is less apt to be expelled. If this apparatus is not

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available, satisfactory results can be obtained by using a three ounce glass aseptic syringe with a plunger attached to a No. 22 F rectal tube. If this syringe is used, a quantity of air should be left in back of the solution, sufficient to insure the emptying of the catheter, but not enough to cause the air to enter the rectum, as this may have a tendency to cause expulsion of the solution.

If sodium amytal is used as the initial dose and labor continues, then four and a half grains of nembutal are given in one hour, followed by the rectal mixture in fifteen minutes or one half hour thereafter. We have found that if labor is definitely established, this procedure can be instituted regardless of the dilation of the cervix or the height of the presenting part. Within three or four minutes from the time of the rectal instillation, the odor of paraldehyde becomes perceptible on the breath of the patient. She quickly falls into a deep sleep, moving from side to side during contractions, sleeping quietly and deeply in the intervals. Labor continues unabated and in the majority of our cases no further medication has been necessary, labor progresses to full dilation of the cervix and the presenting part appears on the perineum. At this stage gas-oxygen is administered and the crowning vertex is lifted over the perineum with prophylactic low forceps. About five or six hours usually elapse before the patients begin to show signs of awakening. At the early stage of labor, a rectal examination is made and if the cervix has been effaced and is four or more fingers dilated and the presenting part is low, the patient can be carried along to delivery with gas-oxygen inhalations during contractions. If the examination shows that labor still has several hours before completion, two to four drams of paraldehyde in one ounce of oil to which has been added one and one half to three grains of nembutal (dissolved in water), is instilled in the rectum. Following this, the patient rapidly falls asleep again and remains so to the completion of labor; gas-oxygen inhalations again being used as the presenting part appears on the perineum. It will be found that small amounts of gas-oxygen are necessary to control the patients. These patients do not resist inhalation anesthesia, thus differing from the other drug combinations where the patients become extremely restless and noisy, attempting to tear the mask from the face. The length of time consumed in the administration of gas-oxygen is considerably less with this method than with the barbiturate-scopolamine group or the straight barbiturate group.

A review of the fifty cases studied in this series shows thirty primiparae and twenty multiparae. The average length of primiparous labors after entrance to the hospital was ten hours and thirty minutes. The average length of multiparous labors was five hours and twenty minutes. The average duration of analgesia during labor for primiparae was nine and one half hours and for multiparae four and one half hours. The average duration of amnesia following delivery was eight hours. Complete amnesia throughout labor was experienced by forty-seven patients. Three patients experienced partial amnesia. There were no failures. Twenty-eight patients showed very slight restlessness, moving slightly with pains. Eighteen patients showed moderate restlessness, turning from side to side and occasionally attempting to sit up with each pain. Four patients showed a degree of restlessness which simulated, but to a lesser degree, that so commonly seen with scopolamine or with the straight barbiturates. These patients, however, were not noisy and did not fight against the inhalation anesthesia. Several patients complained of pain at intervals during labor, but afterwards remembered nothing. There has been no maternal mortality in this series and but one fetal death. This infant presented congenital absence of both eyes and both tibiae and fibulae and died five hours after delivery.

Forty-five babies of this series either breathed or cried immediately following delivery. Five babies required mild resuscitation. Of these, one was a congenital monstrosity, two had tight loops of the cord about the neck which had to be clamped and severed before delivery could be completed. It is fair to assume that this may have contributed to the partial asphyxia. The remaining two babies showed no obvious cause, but responded to mild mouth to mouth resuscitation and carbon dioxide gas inhalations. On several occasions meconium was passed with vertex presentations during labor. However the fetal hearts remained regular and these babies cried readily following the normal completion of labor. The odor of paraldehyde could be detected on the breath of the infants for twenty-four to forty-eight hours after delivery. All the babies of the series nursed normally during this period, with the exception of one, that seemed drowsy for forty-eight hours and nursed poorly. After this period the nursing and weight gain were normal.

Following labor, the majority of the patients continued drowsy and slept in long naps for eight to twelve hours, awakening to take fluid and nourishment and rapidly falling asleep again, thus escaping the usual discomfort of the first twelve hours postpartum.

There were forty-four normal deliveries, which include the use of prophylactic low forceps to the crowning vertex, three mid-forceps deliveries after full dilatation of the cervix, two Scanzoni maneuvers for persistent posterior heads low in the pelvis, with full dilatation of the cervix, and one internal podalic version and extraction.

In several cases where labor lasted ten to twenty-two hours, the rectal administration of

paraldehyde and nembutal was repeated one or more times with resulting excellent amnesia throughout the labor and with no ill effects to the mother or the infants. The following two cases are offered as examples: 1. Case A. R. in a labor lasting twenty-one hours after admission to the hospital received a total of ten and a half grains of nembutal and nine drams of paraldehyde in divided doses. Labor started with an unengaged head and thick cervix and was terminated by a normal delivery. The infant breathed readily. 2. Case A. S. in a labor lasting seventeen hours after admission to the hospital received a total of ten and a half grains of nembutal and ten drams of paraldehyde. This labor started with an unengaged head in a posterior position. The cervix was thick and admitted one finger. She progressed in labor to full dilatation and was delivered by internal podalic version and extraction. This infant breathed as soon as the mouth was brought over the perineum and cried immediately following delivery. The question of cesarian section might have been seriously considered in both of these cases if we had not had available a method of carrying patients through a long, hard labor comfortably and without exhaustion.

At the outset of this series, while attempting to ascertain the optimum amount of paraldehyde, several patients were given as much as 12 drams at a single dose with no undue effects. These patients slept intermittently about thirty-six hours and could be aroused to take nourishment, but quickly fell asleep again. The infants in these cases breathed readily, but remained drowsy for twenty-four hours with no undue effects.

The amount of paraldehyde used in each case varied from four to ten drams, the latter being given in divided doses. The dose of nembutal varied from six to eleven and one half grains, the latter in divided doses.

The average duration of the gas-oxygen administration was one hour and this included the actual delivery and repair. There has been no evidence of any delay in labor. On the contrary, it seems as though there occurs a relaxation of the lower uterine segment with subsequent shortening of the time required to efface and completely dilate the cervix. There has been no evidence of increased postpartum bleeding as a result of this analgesia, and no evidence of rectal irritation or any idiosynerasy to paraldehyde.

We wish to acknowledge our appreciation to the Staff of the New England Hospital for Women and Children, and Katherine Yeo, R.N., Supervisor of the delivery floor, for their kind coöperation.

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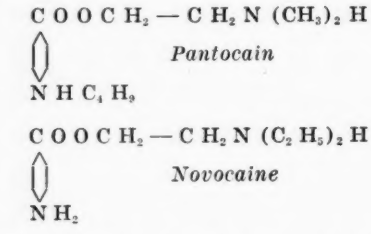
PANTOCAIN IN SPINAL ANESTHESIA*

BY FRANK W. MARVIN, M.D.†

PANTOCAIN is a new anesthetic drug which has been recently synthetized in Germany. Chemically it is of the novocaine series, and is known as Butyl-Amino-Benzoic acid-B-Dimethyl-amino - Ethyl - Ester - Mono-Hydrochloride with the following chemical structural formula in comparison with novocaine:



This new compound exists chemically in the form of small colorless and odorless crystals which are comparatively easily dissolved in water. The resulting aqueous solution has a neutral reaction, and is very resistant to both heat and cold. The solution is stable at room temperature, and no chemical change takes place at winter temperatures below freezing point. Should the solution freeze it will be satisfactory to use after it has thawed out provided the ampoule is not cracked. High temperatures, however, such as prolonged boiling or storage near a radiator may cause decomposition of the solution. It can, however, be easily sterilized. The specific gravity of a one per cent solution of pantocain in an eight-tenths per cent saline solution at twenty-five degrees centigrade is 1.0068, slightly heavier than that of normal spinal fluid which ranges from 1.0012 to 1.0060.

The pharmacological action of pantocain has been carefully investigated from the following three points of view, namely, its toxicity, its optimal dose, and the duration of the effect of the drug. Large doses of pantocain were given intravenously to rabbits before the onset of convulsions; also large doses, that is, many times the intraspinal dose, were given to dogs without any marked alteration in the blood pressure. Hence, pantocain may be considered a safe drug to use in spinal anesthesia. Furthermore, in dogs, intraspinal injections that induced anesthesia of comparatively long duration were followed by complete recovery of the animals used, thus confirming the safety of the drug in these doses. When used intravenously it required at

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least six times the intraspinal dose to produce the lethal effect. The duration of the effect of the drug lasts about two to three times as long as procaine, depending on the size of the dose. In comparison with nupercain (a derivative of quinine) it is just as effective dose for dose, but enjoys the position of being three to four times less toxic. So far as can be determined, both experimentally in animals and clinically in human beings, it is no more toxic than procaine.

Schmidt of Germany was the first to use it clinically. In this country it was used for the first time at the Mayo Clinic on February 20, 1931, since which time they have used it intraspinally in five hundred and eighty-one cases during 1931. We used it at this clinic for the first time on June 26, 1931. It has been given intraspinally here in over one hundred cases without a fatality. Schmidt determined from his work with the drug that the relative toxicity of the effective dose as compared with procaine was one to one. From our clinical experience and that of the others just mentioned, it is reasonable to conclude that we are justified in using this drug intraspinally, and also in placing it in the class of anesthetics which enjoy a fairly wide margin of safety, such as ether and procaine.

The clinical reactions observed in our series of cases were very favorable and warrant the further use of this new drug which has its proper place in spinal anesthesia. Our clinical observations and impressions led us to believe that the blood pressure was not affected by pantocain more than by procaine. A review of all the charts shows that the average systolic drop has been less than that noted with procaine crystals. Twenty-five cases were selected at random, and showed the average systolic pressure to be one hundred and twenty over seventy-two for the diastolic pressure. All of these cases required ninety minutes of operating time. The average fall of the systolic pressure was four millimeters, while that of the diastolic was only two millimeters.

We have other charts in which operations were completed in less than ninety minutes, and also some in which the operative time actually extended to three hours. The pressure readings on these charts were consistent with those listed above.

Case No.	(1) Systolic	(2) Systolic	(3) Diastolic	(4) Diastolic	Operating Time	Operation
156	130	100	80	70	2 hours	Pelvic
251	120	110	76	78	2 hours 30 min.	Double
263	90	80	50	50	2 hours 30 min.	Double
268	120	110	70	60	1 hour 30 min.	Pelvic
272	120	120	78	78	1 hour 45 min.	Pelvic
280	140	140	86	80	1 hour 30 min.	Pelvic
286	120	110	80	70	1 hour 35 min.	Pelvic
287	100	100	60	60	1 hour 45 min.	Periarterial Sympathectomy
321	100	100	60	60	1 hour 30 min.	Double
333	80	90	50	55	1 hour 45 min.	Pelvic
354	100	100	50	50	1 hour 30 min.	Pelvic
403	100	90	60	70	1 hour 30 min.	Ca Prostate
407	130	130	80	80	2 hours	Ca Intestines
408	120	120	70	70	2 hours 35 min.	Double Hernia
414	120	110	80	70	1 hour 30 min.	Recurrent Herniae
436	120	120	70	70	1 hour 30 min.	Pelvic
440	106	106	70	70	1 hour 30 min.	Double
443	100	100	80	80	1 hour 35 min.	Double
444	130	138	80	80	1 hour 30 min.	Plastic and Pelvic
445	118	118	78	78	1 hour 40 min.	Pelvic
446	110	110	70	70	1 hour 45 min.	P. O. Sinus—Patient 96 lbs.
447	110	110	70	70	1 hour 45 min.	Double
452	120	120	80	80	1 hour 45 min.	Ca Rectum
465	135	110	80	80	1 hour 30 min.	Pelvic
470	120	110	80	70	1 hour 30 min.	Pelvic

(1) The systolic pressure reading at the beginning of the operation.
(2) The systolic pressure reading at the completion of the operation.
(3) The diastolic pressure reading at the beginning of the operation.
(4) The diastolic pressure reading at the completion of the operation.

There were some cases in which the blood pressure actually rose. This was probably due to the action of the ephedrin which was used in all cases as a prophylactic measure before the intraspinal injection. The pulse and respirations showed no material changes due to the action of the drug. Since we are beginning to believe more and more (as recently shown by Dr. Nowak at this hospital) that it is the toxicity of the drug which is one of the chief or contributing causes for the fall of blood-pressure, it seems reasonable to believe that if we do not have a marked drop in pressure, our new drug is well within the margin of safety. Apparently no shock, other than that caused by the operation, has been observed in our series of cases in which the drug was used. The blood-pressure readings were carefully recorded every five minutes during the operations, which varied

from thirty minutes to two hours and thirty minutes. Patients were seen later in the day, at intervals from three to six hours, to determine their blood pressures and general reaction to the drug. In practically all cases the pressure was maintained with no marked or serious drop noted or recorded. Should any marked drop in the blood pressure occur, the usual supportive treatment should be instituted at once. We have not yet experienced this condition. If there is nausea, it may be obviated by having the patients properly prepared with drugs; in other words, have them asleep, hence the minimum amount, or entire absence, of nausea.

Respirations have remained quiet and have been maintained at a normal rate while under the influence of the drug. We have not encountered any embarrassment of the respiratory function throughout our series of operative cases. This is probably best explained by the fact that the drug was injected slowly and without much force, so that its anesthetic effect would not interfere with the action of the intercostal and phrenic nerves. It was usually injected in the third lumbar space. We have used it as high as the twelfth dorsal space, but only occasionally. No postoperative pulmonary conditions have arisen after its use in our series. Compared with a series of cases operated under inhalation anesthesia reported by Cutler and Hunt, in which fifty-five pulmonary complications with eleven deaths occurred in one thousand five hundred and sixty-two operations, this type of spinal anesthesia seems preferable for long anesthesias. Nerve injuries have never been noted. Ocular complications and symptoms of meningismus have been entirely absent. This is equally true of meningitis. Postoperative headaches were indeed rare, as only the occasional apprehensive and nervous patient complained of this symptom. The few headaches reported, cleared up quickly after giving a placebo and none of them persisted. Urinary retention has not been encountered although it is frequently observed after inhalation anesthesia.

Our series comprised both females and males, the former outnumbering the latter about two to one. Their ages varied from the youngest, a girl of nineteen, to the oldest, a man of seventy, whereas their weights varied from ninety pounds to one hundred and ninety pounds. There were varied types of operations including gall-bladder, stomach, intestinal (both small and large), epigastric and ventral herniotomies, appendectomies, numerous resections of the different parts of the intestinal tract, and one perforated ulcer of the stomach. Genitourinary operations comprised both kidney and prostatic pathology. In gynecological surgery we did many cases of the so-called double operations where the time element was a factor in the length of the anesthesia. Rectal work including carcinoma was performed a few times. Double inguinal herniotomies also were favorable cases for its use. Bone surgery of the lower extremities, including double-fractured femurs, did well under pantocain. Twenty-five different surgeons operated the above cases without a fatality. Of the total cases operated, twenty per cent were private cases all of which had successful lasting anesthesia. Tests made two to three hours after the initial time of injection showed the anesthesia still persisting to the level of the umbilicus. From our clinical use of this drug, we have shown conclusively that the duration of the effect of the drug lasts about twice as long as procaine. It has not been necessary to add a supplemental anesthetic to complete the operation. This is an added advantage over procaine since the latter does not last quite long enough in some cases.

In regard to the dosage of the drug necessary to produce satisfactory anesthesia, we have had it put up in sterilized sealed ampoules which contain two cubic centimeters of a one per cent solution containing twenty milligrams of the drug. The ampoules hold four cubic centimeters of fluid when filled, and it has been our custom to add two cubic centimeters of spinal fluid to the contents in the ampoule. This will give four cubic centimeters of a one-half of one per cent solution which may be injected intraspinally with the selected dose we wish to use. For example we may inject anywhere from five to twenty milligrams of pantocain as desired from this dilution. Most of the cases have been given twenty milligrams, although in a few we used fifteen milligrams with a very satisfactory and lasting anesthesia. In a few cases we have combined the pantocain with novocaine solution (twenty per cent) for the purpose of making the solution to be injected much heavier than spinal fluid, endeavoring to localize the height of the anesthesia. Doses from sixty up to and including one hundred and twenty milligrams of novocaine, were added to the solution of pantocain before injecting it intraspinally. Anesthesia obtained by this combination did not prove to be any more effective or lasting than that in which our technic called for pantocain alone with spinal fluid which was injected slowly, carefully, and accurately. If we are careful in our technic, we should not experience any trouble or disaster from its use.

CONCLUSIONS

1. Pantocain is a safe and satisfactory drug to use in spinal anesthesia.
2. It produces a long and lasting anesthesia for operations which require from one to two hours.
3. Pantocain is no more toxic than procaine as was shown by its clinical use.

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CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 18341

A CASE OF LYMPHOBLASTOMA

MEDICAL DEPARTMENT

PRESENTATION OF CASE

DR. HUGH MONTGOMERY*: This is the case of a forty-six year old white married Canadian housewife who was first seen in the skin clinic of another hospital in August, 1931, because of an eruption on her abdomen. In September she was seen in the cancer clinic of the same hospital, where an oblong raised patch of erythema measuring about 8 by 5 centimeters was found below the navel, with moderate induration and some nodules scattered below the surface. When this had first been noticed that spring it was the size of a half dollar. It had itched, but otherwise had not troubled her.

In September she went to the laboratory of a second hospital, where the lesion was removed. The pathologic report was lymphoblastoma.

She was then seen at the ear, nose and throat clinic of the first hospital because of nasal obstruction, and in October, 1931, a section was removed from the inferior turbinate for biopsy. The report on this was the same as that on the section removed from the abdomen. At the same time a diagnosis of rheumatic heart disease was made in the medical department of that hospital. The signs that were found in the heart at that time are not mentioned in the report we have received.

On March 22 she entered the Eye and Ear Infirmary complaining of nasal obstruction, but because of vaginal bleeding of two weeks' duration was transferred to the Massachusetts General Hospital the following day.

First admission. On March 23 she was first admitted to this hospital. She had lost 20 pounds' weight in the preceding eight months. In the interim since the previous work done at the other hospital she had had some headaches, nasal obstruction and recently moderate dyspnea. Her bowels were chronically constipated. Five months before that time she had an attack of back and leg pain lasting two weeks, then passing off.

Examination was negative except for a lacerated cervix. The white blood cell count was 15,000, the hemoglobin 65 per cent.

Dilatation and curettage was done. On March 30 she was discharged to the Eye and Ear Infirmary for treatment of nasal obstruction.

History of interval. A tumor of the nose and nasopharynx was removed at the Eye and Ear Infirmary. The pathologic report was again lymphoblastoma, probably lymphosarcoma. She was given a short course of x-ray treatment. Routine blood examination at that time was roughly normal.

Second admission. On May 5 of this year she entered the Massachusetts General Hospital complaining of weakness, very recent icterus and clay colored stools, some nausea but no vomiting except when it was self induced because she thought it made her feel more comfortable. She weighed 160 pounds a year ago and now weighed 132.

Physical examination showed her to be a thin woman with moderate jaundice. There was an irregular mass in the upper abdomen 4 centimeters below the xiphoid in the midline, the larger portion on the right, a small portion on the left.

The routine blood examination was normal except for slight achromia. The bleeding time and clotting time were normal. The van den Bergh on the blood was 11.7 to 16.5 milligrams, direct reaction. Five readings were done. A liver function test showed 100 per cent retention. The sedimentation rate was accelerated. A duodenal tube was passed and left down all night. No bile was obtained, gross or by chemical test.

She was given a course of x-ray treatment. The jaundice seemed to clear. At the end of two weeks she was discharged. The same diagnosis was made as previously.

History of interval. A week after her discharge, two weeks before her third admission, she became nauseated and vomited. The nausea persisted. On the fourth day she became jaundiced and began to vomit bile. Six days before admission she passed coal black stools and had small bluish red purpuric spots all over her body and upper abdominal tenderness which persisted. She had persistent vomiting for eight days before admission. For seven days she had been vomiting pure bile or else retching without bringing up anything.

Third admission. On June 10 she was admitted with a complaint of weakness and dyspnea.

Physical examination showed a very thin, weak woman with a greenish yellow color There were multiple purpuric spots, some as large as a quarter dollar, some very small, some confluent. She had a tender mass in the epigastrium about 10 centimeters below the xiphoid, irregular in outline, evidently larger than it had been on examination by the surgeons in May.

Blood examination showed a white count of 16,900 with a questionable 32 per cent myelocyte count on one occasion, 23 per cent on another. The red cell count was 4,670,000. The

*Recently senior interne on the East Medical service.

hemoglobin was 85 per cent at admission, 75 per cent at the next examination four days later. The red cells were normal in size, shape and color. The platelets seemed to be diminished.

Our impression was that she had lymphoblastoma involving the glands of the upper abdomen, and we wondered because of the finding of myelocytes whether she did not have now a change due to a leukemic process.

The urine at that time was roughly negative except for the presence of a large amount of bile. The specific gravity of both specimens taken was 1.010. We do not know whether she could concentrate beyond that or not. The nonprotein nitrogen was 50 milligrams. A liver function test showed 100 per cent retention. The blood serum was green, so we could not read the icteric index against the standard. The van den Bergh was 36.22 milligrams per 100 cubic centimeters, direct reaction.

She was in very poor condition when she entered. As she was very dehydrated she was given daily infusions of glucose. She failed rapidly. The chart showed a low fever. On the ninth day she very quietly died.

A PHYSICIAN: Was the spleen palpable?

DR. MONTGOMERY: We did not know whether the spleen was involved or not; we thought not.

CLINICAL DISCUSSION

DR. TRACY B. MALLORY: This of course might have been considerably more of a diagnostic problem were it not for the peripheral manifestation on the skin with which it started and which led to biopsy. There can be no doubt that the case is some form of lymphoblastoma, the differential point being what type.

DR. WYMAN RICHARDSON: It is interesting to consider the question of anemia in its relation to tumors. Some lymphoblastomas apparently produce quite severe anemia which it is almost impossible to explain on the basis of bone marrow invasion alone. Radiation of local tumor masses often causes the anemia to improve too. I have no idea why that is. It is an interesting thing to think about.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Lymphoblastoma.

ANATOMIC DIAGNOSIS

Lymphatic leukemia, aleukemic stage.

PATHOLOGIC DISCUSSION

DR. MALLORY: Autopsy on this case showed a marked enlargement of the liver and spleen, the liver weighing 2400 grams and the spleen 800 grams, so that at least part of the mass felt was certainly the spleen. The kidneys were not greatly enlarged, but were of a quite unusual grayish pink color and showed unmistakable gross evidence of invasion by the tumor process. The majority of the lymph nodes were enlarged. The bone marrow appeared grossly negative.

The sections I think are of the greatest interest. Even with the low power it is very obvious that in the liver one has massive infiltration of white cells all of which appear to be of the lymphocyte series and moderately well differentiated. The infiltration is most marked in the portal areas, but slighter degrees of infiltration run well towards the center of the lobules along the sinuses. The type of infiltration is exactly similar to that seen in the average case of leukemia. The spleen showed essentially a similar process.

In the enlarged lymph nodes the architecture is not destroyed, but there is a diffuse hyperplasia throughout the nodes and lymphocytes are being formed throughout the pulp. There are no true germinal centers left. Also the capsule of the node is invaded by the process and there is a diffuse invasion of the surrounding fat tissue.

The kidney shows very marked infiltration, very much like that of the liver, though here it is a little more focal, not quite so diffuse.

The bone marrow from several areas of the sternum and the vertebrae shows essentially the same picture. In the long bones the bone marrow was fatty. In the vertebrae the proportion of fat cells is considerably diminished. There are more marrow cells than there should be. All types of marrow cells can be found and in addition there is a considerable number of cells which appear to be lymphocytes. There is a moderate diffuse infiltration of the marrow by the same type of cells that we found in the liver and spleen. It has not got to the state of really replacing the marrow elements as often happens in the later stages of true leukemia.

My feeling on the case would be that with as diffuse an infiltration of nearly all the organs of the body as we have here, without the formation of any discrete tumors anywhere, we should have to classify the case as aleukemic leukemia, the strongest points against that being the lack of anemia and the relatively slight bone marrow involvement.

CASE 18342

RAPIDLY FATAL ILLNESS CHARACTERIZED BY TOXICITY AND DIARRHEA

CHILDREN'S MEDICAL DEPARTMENT

PRESENTATION OF CASE

DR. RALPH W. DAFFINEE*: A four year old boy was admitted to the hospital October 4 at noon. The complaint was that he had become very sick during the past twenty-four hours with headache, drowsiness and loose bowel movements.

The family came to Boston from England two years ago. A sister, aged three, had also had loose bowel movements for one day on October 3 but had not been sick. The other members of the family were perfectly well.

*Recently resident on the Children's Medical service.

The patient had had measles and whooping cough. Otherwise he had always been a healthy, active child. He ate well, but refused to drink milk. He had never had a serious gastro-intestinal disturbance.

He was perfectly well until five p. m. on October 3. Then he complained of headache. At six o'clock he had a loose bowel movement and seemed feverish. He was given "ex-lax". He vomited once at ten o'clock. That night he had five or six bowel movements. His condition seemed somewhat similar to that of his sister. The parents were not alarmed until the next morning, when he became very stuporous and had a convulsion. A physician was called and referred the child to the hospital.

The child appeared prostrated and semi-comatose. He mildly resisted examination. He was pale and slightly cyanotic. The skin and mucous membranes of the mouth appeared dry. There was no eruption. His breathing was shallow. The pupils reacted sluggishly. A film was forming on the corneae. There was slight tenderness over the epigastrium. The liver and the spleen were not felt. There was some stiffness of the neck. Kernig's sign was positive.

While in the hospital he had two bowel movements, small pasty green and white stools with some mucus but not watery or bloody. Cultures of these were taken and were reported ten days later.

Blood examination showed 10,400 white cells, 59 per cent polymorphonuclears, 40 per cent lymphocytes, hemoglobin 90 per cent, 6,860,000 red cells.

The temperature at admission was 104°, the pulse 156. The respirations were 26.

The child was dehydrated; therefore 600 cubic centimeters of normal saline with 2½ per cent glucose was given by hypodermoclysis. The child was obviously toxic. He became gradually worse. The temperature rose to 105.4°. The heart rate increased to 240. Transfusion was decided upon, but before it was done the child died, at seven p. m., twenty-six hours after the onset of the illness.

On the day of the patient's death, another sister aged one year and a half developed diarrhea. The two sisters were admitted to the hospital but were not particularly ill while there. They were followed for a long time afterwards in the Outpatient Department. The parents felt that they were not really well until a year after the illness. The mother and father had no symptoms at the time.

Clinical Discussion

Dr. Harold L. Higgins: The clinical diagnosis in this case was dysentery. We based our diagnosis on the toxicity of the patient, on the fact that he had loose green stools with mucus and the fact that he had a sister with the same condition.

There are several things one had to consider in the differential diagnosis. One was arsenic poisoning; another was meningitis; another was an enteric infection caused by one of the organisms of the paratyphoid group.

It is usual with dysentery for more than one member of the family to have the disease. Here we have the story of two other children also having diarrhea. It is also not infrequent, when more than one person in the same family have dysentery, for one to have a very mild attack and another to be very ill and very toxic. The exact reason for that we do not know with certainty.

The death of patients with dysentery may usually be traced to one of four causes. They may die of toxicity, that is from the poisons of the dysentery bacillus which act upon some of the vital cells and destroy them; or they may die of the effect of dehydration, loss of fluid; or they may die of inanition, starvation; or they may die from some secondary infection, as pneumonia. In this case the toxicity was predominant and out of proportion to the intestinal symptoms. I feel that the neurological signs here probably can be explained on the basis of a toxic encephalitis caused by the toxin of the dysentery bacillus.

This child did show some dehydration; the red count was as high as 6,860,000. For that reason fluids were forced. It was felt advisable to give him fluids in other ways than by mouth. I do not know whether he was able to take water by mouth or not. Can you inform us about that?

Dr. Daffinee: He did not take it very well.

Dr. Higgins: Fluid was given by hypodermoclysis. Inanition death from dysentery occurs weeks or months after the onset of the diarrhea and was not a factor in this case. The toxicity in dysentery is best combated by transfusion. Transfusion many times seems to furnish antitoxic principles that are in the donor and to lead to improvement. A transfusion had been decided on in this case, but the child died before the matching of his blood with the donor's was complete.

The stools in dysentery are usually characterized by mucus and blood. They usually are not so watery as the stools of intestinal indigestion or cholera or paratyphoid. That is, dysentery is mainly a colitis or an ileocolitis rather than an enteritis. The ordinary food poisonings are usually characterized by enteritis and therefore by watery rather than mucous stools. The fact that the stools in this case had a good deal of mucus in them rather pointed towards irritation in the large bowel where the mucous glands are prominent, rather than to trouble in the small bowel, which would lead to rapid evacuation of their contents and watery stools.

Our diagnosis in this case was bacillary rather than amebic dysentery because that is the com-

moner type of dysentery here in the North. Amebic dysentery is seldom seen in Boston, and the child had not been living in the South. He came from England two years previously, but there is no previous history of his having been ill with diarrhea.

I do not believe that the milk was responsible for the infection in this case. The boy did not drink milk, so that source is ruled out. Dysentery ordinarily spreads through feces, fingers and flies, the three F's. I presume that the bacillus must have got into the food, as some other members of the family developed diarrhea. Somebody in the family may have been a carrier.

CLINICAL DIAGNOSIS (FROM HOSPITAL REPORT)

Bacillary dysentery.

ANATOMIC DIAGNOSES

Dysentery, Flexner's type.
Status lymphaticus.
Septicemia (?) streptococcus viridans.

PATHOLOGIC DISCUSSION

DR. JOHN I. BRADLEY: The striking thing about the autopsy was the paucity of findings. The autopsy was grossly almost negative. The small intestine showed only injection, no ulceration. The one rather prominent feature was the presence of enlarged mesenteric and retroperitoneal lymph nodes and marked prominence of the Peyer's patches in the small intestine. The bronchial glands, however, were not enlarged. The thymus weighed 40 grams. That was regarded as probably representing slight enlargement. The brain in gross was negative. Microscopically there was dilatation of the smaller vessels in a number of areas. A few red blood cells were seen in the perivascular spaces of the subependymal vessels, but that was all. The autopsy was done fourteen and one quarter hours after death. Blood cultures showed streptococcus viridans. In cultures of the stool during life the bacillus dysenteriae, Flexner's type, had been isolated.

Dr. Dienes, would it be reasonable to expect such a short course and such marked toxicity with this organism?

DR. LOUIS DIENES: Yes. In general the Shiga type of dysentery bacillus is more toxic than Flexner's type, but on the other hand there are cases in which the clinical symptoms are just as severe in infections caused by the Flexner as by the Shiga type of bacillus. The finding of Flexner's type of bacillus is not absolute proof that the disease was caused by this type of bacillus, because we find a certain number of cases in which the stools show organisms of that type, but which die of other diseases. This is only a possibility, but a possibility which has to be kept in mind.

DR. BRADLEY: How much importance should be attributed to the finding of streptococcus viridans in the blood cultures fourteen hours after death?

DR. DIENES: Very little I think, because the finding of non-hemolytic streptococcus in the blood at autopsy is not rare.

DR. HIGGINS: Dr. Mallory has several times directed my attention to a finding that children who die very quickly of some acute illness like dysentery or pneumonia have enlarged lymph glands and an enlarged thymus. It would seem that there might be a possibility that some condition analogous to status lymphaticus may have been a contributing factor in the speedy termination in this case.

DR. BRADLEY: It certainly has to be considered, Dr. Higgins. Without question there was an unusual and abnormal prominence or hyperplasia of the lymphoid tissue here. The difficulty is to distinguish cause and effect, cart from horse. The British commission for investigation of that question reviewed the coroners' cases of sudden death and came to the conclusion that there is no definite proof that thymic persistence with the other stigmata of "status lymphaticus" such as lymphoid hyperplasia and aortic hypoplasia is a sole cause of death. Nevertheless in children dying rather suddenly we do find large thymuses and enlargement or hyperplasia of the lymphoid tissue in a sufficient number of cases to make one still loath to give up entirely the idea that they may be of contributory significance.

A PHYSICIAN: Was this a large, fatty type of child?

DR. DAFFINEE: As I remember him he was rather large boned and rather fair. He certainly was not oversized.

DR. BRADLEY: Our record describes him as well developed and nourished, with fairly marked pallor of the mucous membranes.

A PHYSICIAN: Did they get in the hospital the same type of organism from the other children in the family?

DR. BRADLEY: Yes. They had positive cultures from the stools of all three children, and cross agglutinations between organisms recovered from the stools of this patient who came to autopsy and the sera of his sisters were positive, indicating that the same organism was responsible for all three cases.

DR. TRACY B. MALLORY: Fatal dysentery is relatively rare in Boston in my experience and almost wholly confined to very young children. The strains involved are varied, the Flexner type being the commonest over a period of years, though occasional epidemics of other strains such as Sonné are seen from time to time. The very toxic Shiga type is the least common in this locality. The postmortem findings may not be striking, in fact I have more than once missed the diagnosis on the gross examination and only the cultural findings have made clear the cause of death.

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STERILE SURGICAL CATGUT

THERE is probably no one material used in surgery more generally accepted as being sterile, but in reality more liable to be contaminated, than catgut. Meleney and Chatfield[1] found that 22 out of 174 specimens, submitted by 12 surgical clinics in 11 different states, yielded growths of spore-bearing bacilli. In Germany, a relatively recent paper[2] recorded 80 per cent of contaminations. Bulloch, Lampitt and Bushill[3], in England, found frequent contaminations in tubes purchased in the open market and recommended a method of testing for sterility, which the British government has since made a legal requirement for all catgut manufacturers.

From the source of the material, it is obvious that raw catgut must be grossly contaminated. The bacteria present include not only vegetative forms of all sorts which are easily killed during the process of manufacture of surgical catgut, but also spores of the spore-forming bacilli which are very resistant. While it is true that the majority of bacilli cultured from non-sterile surgical catgut are usually considered to be non-pathogenic, instances have been reported in which tetanus bacilli[4] and bacilli of the gas-gangrene group[5] have been isolated.

The non-sterility of surgical catgut results, in many instances, from inadequate methods used by the manufacturers to test for sterility. The importance of preliminary washing in such tests to neutralize chemically the bacteriostatic compounds in the tubing solutions, first advised by Geppert[6] and later by Bulloch, Lampitt and Bushill[3] and Meleney and Chatfield[1], is not generally appreciated. Furthermore, an anerobic method of culturing, suitable for the growth of the more fastidious anerobic bacilli, must be employed[1]. *Control of sterility should be legally placed under government supervision,* as in England. The National Institute of Health in Washington, which so efficiently guards the potency and sterility of therapeutic sera and certain other biologic products distributed outside the State of manufacture, is the logical agency for control.

Until supervision is authorized, every surgeon should attempt to use catgut which is released for distribution only after proper and efficient sterility tests. The use of "boilable" catgut is not a remedy, as a temperature of 100°C. for a short period of time has little or no effect on spores, particularly in a medium devoid of water, such as is necessary for the tubing fluid with this type of suture. Lacking assurance that the manufacturer releases surgical catgut only after a sterility test such as that recommended by Meleney and Chatfield[1], all large hospitals should regularly test their catgut for sterility; and, hospitals and individual surgeons that lack the necessary facilities should both turn to them for advice.

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Dr. Dienes, would it be reasonable to expect such a short course and such marked toxicity with this organism?

DR. LOUIS DIENES: Yes. In general the Shiga type of dysentery bacillus is more toxic than Flexner's type, but on the other hand there are cases in which the clinical symptoms are just as severe in infections caused by the Flexner as by the Shiga type of bacillus. The finding of Flexner's type of bacillus is not absolute proof that the disease was caused by this type of bacillus, because we find a certain number of cases in which the stools show organisms of that type, but which die of other diseases. This is only a possibility, but a possibility which has to be kept in mind.

DR. BRADLEY: How much importance should be attributed to the finding of streptococcus viridans in the blood cultures fourteen hours after death?

DR. DIENES: Very little I think, because the finding of non-hemolytic streptococcus in the blood at autopsy is not rare.

DR. HIGGINS: Dr. Mallory has several times directed my attention to a finding that children who die very quickly of some acute illness like dysentery or pneumonia have enlarged lymph glands and an enlarged thymus. It would seem that there might be a possibility that some condition analogous to status lymphaticus may have been a contributing factor in the speedy termination in this case.

DR. BRADLEY: It certainly has to be considered, Dr. Higgins. Without question there was an unusual and abnormal prominence or hyperplasia of the lymphoid tissue here. The difficulty is to distinguish cause and effect, cart from horse. The British commission for investigation of that question reviewed the coroners' cases of sudden death and came to the conclusion that there is no definite proof that thymic persistence with the other stigmata of "status lymphaticus" such as lymphoid hyperplasia and aortic hypoplasia is a sole cause of death. Nevertheless in children dying rather suddenly we do find large thymuses and enlargement or hyperplasia of the lymphoid tissue in a sufficient number of cases to make one still loath to give up entirely the idea that they may be of contributory significance.

A PHYSICIAN: Was this a large, fatty type of child?

DR. DAFFINEE: As I remember him he was rather large boned and rather fair. He certainly was not oversized.

DR. BRADLEY: Our record describes him as well developed and nourished, with fairly marked pallor of the mucous membranes.

A PHYSICIAN: Did they get in the hospital the same type of organism from the other children in the family?

DR. BRADLEY: Yes. They had positive cultures from the stools of all three children, and cross agglutinations between organisms recovered from the stools of this patient who came to autopsy and the sera of his sisters were positive, indicating that the same organism was responsible for all three cases.

DR. TRACY B. MALLORY: Fatal dysentery is relatively rare in Boston in my experience and almost wholly confined to very young children. The strains involved are varied, the Flexner type being the commonest over a period of years, though occasional epidemics of other strains such as Sonné are seen from time to time. The very toxic Shiga type is the least common in this locality. The postmortem findings may not be striking, in fact I have more than once missed the diagnosis on the gross examination and only the cultural findings have made clear the cause of death.

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STERILE SURGICAL CATGUT

THERE is probably no one material used in surgery more generally accepted as being sterile, but in reality more liable to be contaminated, than catgut. Meleney and Chatfield[1] found that 22 out of 174 specimens, submitted by 12 surgical clinics in 11 different states, yielded growths of spore-bearing bacilli. In Germany, a relatively recent paper[2] recorded 80 per cent of contaminations. Bulloch, Lampitt and Bushill[3], in England, found frequent contaminations in tubes purchased in the open market and recommended a method of testing for sterility, which the British government has since made a legal requirement for all catgut manufacturers.

From the source of the material, it is obvious that raw catgut must be grossly contaminated. The bacteria present include not only vegetative forms of all sorts which are easily killed during the process of manufacture of surgical catgut, but also spores of the spore-forming bacilli which are very resistant. While it is true that the majority of bacilli cultured from non-sterile surgical catgut are usually considered to be non-pathogenic, instances have been reported in which tetanus bacilli[4] and bacilli of the gas-gangrene group[5] have been isolated.

The non-sterility of surgical catgut results, in many instances, from inadequate methods used by the manufacturers to test for sterility. The importance of preliminary washing in such tests to neutralize chemically the bacteriostatic compounds in the tubing solutions, first advised by Geppert[6] and later by Bulloch, Lampitt and Bushill[3] and Meleney and Chatfield[1], is not generally appreciated. Furthermore, an anerobic method of culturing, suitable for the growth of the more fastidious anerobic bacilli, must be employed[1]. *Control of sterility should be legally placed under government supervision,* as in England. The National Institute of Health in Washington, which so efficiently guards the potency and sterility of therapeutic sera and certain other biologic products distributed outside the State of manufacture, is the logical agency for control.

Until supervision is authorized, every surgeon should attempt to use catgut which is released for distribution only after proper and efficient sterility tests. The use of "boilable" catgut is not a remedy, as a temperature of 100°C. for a short period of time has little or no effect on spores, particularly in a medium devoid of water, such as is necessary for the tubing fluid with this type of suture. Lacking assurance that the manufacturer releases surgical catgut only after a sterility test such as that recommended by Meleney and Chatfield[1], all large hospitals should regularly test their catgut for sterility; and, hospitals and individual surgeons that lack the necessary facilities should both turn to them for advice.

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SHALL MASSACHUSETTS LEGALIZE CHIROPRACTIC?

For several years attempts have been made to secure the enactment of one bill after another before the Legislature, sponsored and advocated by Chiropractors and their friends. The Joint Legislative Committee, representing the Massachusetts Medical Society and the Massachusetts Homeopathic Medical Society and other physicians, have met the effort with plain, clear and logical statements and arguments which have secured sufficient support to prevent the enactment of any of these bills. This movement on the part of the chiropractors has been prosecuted with persistent intensity, but hitherto without avail although endorsed by many lay people.

There has been so much publicity concerning this movement that a very considerable amount of interest has been created throughout the State respecting this appeal for state recognition both for and against.

Not content with the adverse decision by the Legislature, the proponents of the several bills to recognize this cult have decided to put the question before the voters of this Commonwealth under the resource of "An Initiative Petition." A description of the proposed law is as follows as it appears in the Petition*:

"The proposed law defines and legalizes the practice of chiropractic; establishes a board of registration of chiropractors and defines the number, mode of appointment, tenure, qualification and compensation of its members, and its powers and duties; provides for the preliminary qualifications, examination, certification, original registration and annual license of chiropractors and regulation of their practice, and defines the mode, subjects of and fees for examination, the mode of practice and penalties; and other matters related thereto, which are chiefly as follows:

"That chiropractic be 'the science or practice of locating and adjusting by hand the malpositions of the articulations of the human spine.'

"That any person, with one preceding year's residence in this Commonwealth and with degree from a qualified chiropractic school received at least one year prior to the date when this law takes effect, may, within ninety days after such date, be certified by the board at its discretion without examination.

"That otherwise no person, except a member of the board of registration, shall practice chiropractic unless, being over twenty-one years of age, of good moral character and graduated from a reputable chiropractic school after personally attending courses of at least twenty-two hundred sixty-minute daylight hours given to persons qualified to graduate from a public high school, he shall qualify by examination, pay a fee of twenty-five dollars and be registered and certified; except that any person licensed in another State maintaining standards equal to those of this Commonwealth may be registered without examination.

"That the examination be scientific and practical in character, in subjects including anatomy, physiology, symptomatology, hygiene, sanitation, chemistry, histology, pathology, chiropractic analysis, and the principles and practice of chiropractic.

"That the board may, after hearing, revoke a certificate for cause.

"That the board shall make an annual report and shall keep public records of its proceedings and of the names of persons examined or registered by it and of the names and addresses of all registered chiropractors, who shall pay an annual license fee, promptly notify the board of any change in address and furnish such other information as the board may require.

"That the board consist of three members, residents of the Commonwealth for three years, graduates of a chartered chiropractic school having power to confer degrees in chiropractic, to be appointed by the governor, with the advice and consent of the council, for a term of three years.

"That the board have authority to administer oaths, summon witnesses and take testimony and adopt reasonable rules and regulations pertaining to their duties, and that they pay into the state treasury fees received by them, and that they receive compensation and reimbursement for expenses in such amounts as may be annually appropriated therefor not exceeding the amount of such fees.

"Penalties are provided for practicing chiropractic without holding a certificate or for using the words "Chiropractic Practitioner", the letters "D.C.", or any title or letters indicating engagement in such practice, or for buying, selling or fraudulently obtaining any diploma or record of registration.

"It is further provided that the present law relating to the registration of physicians and surgeons and providing penalties for the unauthorized practice of medicine shall not be held to discriminate against registered chiropractors; but no chiropractor, unless registered as a physician or surgeon, may practice obstetrics, or administer drugs, or perform surgical operations by the use of instruments, or hold himself out as and for other than a chiropractor."

All legal requirements seem to have been complied with and the question of approval or dissent will be before the voters of this State at the November election.

With this broader approach there are definite uncertainties respecting the wishes of the State at large.

Preceding activities by proponents and oppo-

*A circular of information respecting this Initiative Petition will be sent by the Secretary of State to all registered voters one week before election.

nents stand now as minor skirmishes which precede the major engagement.

The proponents will employ arguments designed to show that Chiropractic is a valuable therapeutic resource which has been neglected by physicians, citing in support testimonials of persons who believe that certain ailments have been cured thereby. This has been one of the means which has been effective in arousing enthusiastic approval.

The other important and appealing claim will be founded on the alleged intrenched position of organized medicine which strives to prevent the advent of unrecognized groups into the practice of the healing art. With these two major arguments several minor supporting suggestions will be used.

Both claims are fallacious. Chiropractic like most of the cults has never made a valuable contribution to pathology, preventive medicine or public health procedures. It simply claims to adjust displaced structures and aside from the use of drugs, the employment of surgical operation with instruments and the practice of obstetrics seeks to secure most of the power and authority conferred by law on educated physicians.

The assumption that practitioners of the theory of Chiropractic have knowledge and technic comparable with our orthopedists and neurologists is untenable. Many people, however, believe that they have suffered with diseases unrecognized and uncured by physicians, but from which recovery has taken place while under chiropractic treatment.

Testimony of this kind is convincing with many because the facts relating to functional disturbances, organic diseases and the inherent recuperative power of the human machine are unknown to a large proportion of mankind.

Another very important factor entering into this problem is the lack of knowledge by the laity of the wonderful accomplishments of scientific medicine and the multitude of scientifically trained men now engaged in efforts to solve the mysteries of disease and devise appropriate healing resources.

To compare Chiropractic with regular medicine is one of the paradoxical resources of those who feel that regular medicine has failed to take cognizance of some serious ills.

The part to be played by the medical profession in this contest must include an awakening of understanding of the great service now rendered by scientific practice and the expectation of further progress through research.

Dr. Thomas J. O'Brien's arguments*, as presented before the Plymouth District Medical Society, July 21, 1932, should be examined for full understanding of the matter before the people of this State.

*See page 284, issue of August 11, 1932.

EDWARD WYLLYS TAYLOR, M.D.

EDWARD WYLLYS TAYLOR, M.D.

In the death of Edward Wyllys Taylor, August 17, 1932 after an illness of many months' duration, American neurology loses one of its outstanding figures and Boston one of its most esteemed practitioners and teachers. Few physicians have been more respected or admired; as an instructor in the difficult technique of neurological diagnosis to a generation or more of students, he will be long remembered. His patients, also, quickly grasping his essential kindness, came to him repeatedly for advice; physicians, knowing his sound knowledge of nervous disease, depended upon him in consultation.

Born in Montclair, New Jersey, May 7, 1866, the son of Alfred and Jane Brown Tucker Taylor, he attended public schools and entered Harvard College in 1884. During his four undergraduate years he made many friends, particularly a number of men who later practiced medicine in Boston. He made philosophy his major subject, received honorable mention as a student, and wrote a dissertation for his commencement in 1888. He entered the Harvard Medical School the next year and obtained the Boylston Medical Society prize in February, 1891, with an essay entitled, "The Mental Element in the Treatment of Disease", published the same year.

After his graduation he went back to his home and then left for study in Europe, spending the time from October, 1891 until September, 1893 in Germany, at Berlin and Freiburg. He became a student of Professor Hermann Oppenheim in Berlin and showed so much promise that he was appointed assistant in Professor Oppenheim's department, serving there from September, 1892 until August, 1893. During this period he was largely concerned with the anatomy and pathology of the nervous system. There were at that time no laboratories devoted to neuropathology in this country and relatively few in Europe. It was, therefore, a rare opportunity to study under the leading clinical

neurologist in Europe. The stains then used in the preparation of sections were less elaborate than at present, although several of them at least have persisted unchanged. The methods of staining particularly in vogue then were those of Weigert and a modification by Pal, in which the gray matter was completely decolorized. These stains were, and are, invaluable in determining myelin degeneration and in tracing fibre tracts both normal and degenerated. Golgi's silver preparations were just appearing and there were then no adequate means of staining the neuroglia, about which, in fact, little was known. The ample material at one's disposal in Oppenheim's laboratory and the close association which he always maintained between clinical manifestations and pathological changes constituted valuable material for an ardent student.

In the autumn of 1893 he began the practice of neurology in Boston and soon became associated with the Harvard Medical School. As the result of his work with Oppenheim, he had become interested in the pathology of the nervous system and his first connection with the school was in this department, under Professor W. T. Councilman. On his first visit to Dr. Councilman, he showed him some very large and very beautiful slides of the base of the brain which he had cut and stained in Oppenheim's clinic. Such slides had never been seen here before and did much to recommend Dr. Taylor for the school position he sought. A small laboratory was established and from this beginning grew one of the first, if not the first, laboratory entirely devoted to neuropathology, which later expanded into a large department with a much widened scope. In those early days the nervous system in its clinical and pathological aspects had small recognition at the medical schools. It was, perhaps, desirable that the later developments should, at this period, have had as a foundation some knowledge of the pathological changes underlying clinical symptoms. Previous to that time there had been no university recognition of the existence of such a subject as neuropathology, in spite of J. J. Putnam's efforts in its behalf.

Later, he entered the department of nervous disease and began his teaching career, which lasted until he retired in 1926. On the resignation of Dr. Putnam in 1905 he became the James Jackson Putnam Professor of Neurology, Emeritus in 1926. During most of this period he carried a large part of the burden of the neurological teaching at the Harvard Medical School, using the out-patient department at the Massachusetts General Hospital as his main source of clinical material. As a teacher he was especially popular with the students. Easy of speech, quick to draw out the essential facts from a patient, often humorous, and with a remarkable ability to draw rapid diagrams on the blackboard, Taylor's clinics were always well attended and frequently applauded. His clearness and simplicity also appealed to many graduate students in his successive years as "the chief" at the Massachusetts General Hospital. His enthusiasm for neurology was strongly appreciated by Dr. Harvey Cushing, a "house pupil" 1896-1897; they worked together, both dissatisfied with neurosurgery as it was practiced at the time. It is probable that Dr. Taylor's influence on the younger man was not inconsiderable.

At the Massachusetts General Hospital he was an enthusiastic advocate for the importance of his department and it was a pleasure for him to watch it grow from a small out-patient group to a well-established unit, with its own ward in the general hospital and with multiple ramifications into the other departments. He had a great influence in training younger men, particularly Dr. James B. Ayer, who was responsible for the founding of the spinal fluid laboratory.

Dr. Taylor established many connections with hospitals in cities near Boston. One of his particular interests was the Long Island Hospital, a city institution for chronic disease in Boston Harbor. Here he did his earliest pathological work after returning from Berlin. Up to the time of his death, he served as secretary of the Board of Visiting Physicians. In his earlier days he did much of his teaching at this institution. Some of his best papers resulted from long clinical observation of chronic cases with correlated necropsy reports.

In practice he soon gained the respect of his brother physicians. His friendly spirit endeared him to many. This was clearly shown by the unusual spontaneous enthusiasm of his medical colleagues at his sixtieth birthday, May 7, 1926, when a complimentary dinner, largely attended, was given in his honor. (*Boston M. and S. J.* 194:955, 1926).

From the time of his graduation from college he was always interested in the literary side of medicine. He served on the staff of the *Boston Medical and Surgical Journal* as assistant editor (1899-1912) and finally as editor (1912-1914 inclusive). Other activities included positions on the editorial board of the *Archives of Neurology and Psychiatry*, the *Journal of Nervous and Mental Disease*, and the *New England Quarterly*. For many years he edited the *Collected Reprints, Department of Neurology, Harvard Medical School.*

Dr. Taylor became a member of the American Neurological Association in 1895, vice-president in 1912, president in 1917, and councillor from 1918 to 1922. He was a member of many societies, serving as secretary of the Boston Society of Medical Sciences; president of the Boston Society of Psychiatry and Neurology in 1913; president of the Boston Society for Medical Improvement in 1912-13; president of the Boylston Medical Society in 1911-12, chairman of the

Medical Section of the Suffolk County Medical Society; and vice-president of the Association for Research in Nervous and Mental Disease in 1921. Other memberships included the American Academy of Arts and Sciences, the American Medical Association, the American Psychopathological Association, the Association for the Study of Internal Secretions, the Massachusetts Medical Society, the New England Psychiatric Society, and the American Association for the Advancement of Science. From 1899 to 1901 he served, also, as Professor of Neurology at the University of Vermont. He was granted an A.M. degree by Harvard University in 1899.

His medical contributions consisted of nearly one hundred papers, many of them, before publication, being read before local or national societies. Particular interest was shown along two lines of thought: multiple sclerosis which occupied his attention from the time of his earliest medical work in Oppenheim's clinic up to the latter years of his life, and psychotherapy, which was always prominent in his mind. In 1894 he published, in German, a paper on the pathology of multiple sclerosis. This was the beginning of his interest in the disease; a second paper, in 1906, contained some of the best illustrations of pathological tissues published in this country up to that date; another on the location of lesions with respect to symptoms was presented at a meeting of the Association for Research in Nervous and Mental Diseases, 1921, and chapters on the same subject were contributed to *Nelson's Loose-Leaf Living Medicine,* 1920 and *Cecil's Text-Book of Medicine,* 1927.

In 1906, psychotherapy in its newer applications was considered, and, the next year, the whole subject was reviewed. Strongly of the opinion that simple methods have a place in psychotherapy, he published his views from time to time. These papers, published during the height of the Freudian controversy, served a double purpose, for they not only tended to draw together the extremists, but served to emphasize the importance of psychotherapy in the minds of the medical practitioner at a time when such clear-cut, logical exposition was greatly needed. "My only present contention," he wrote in 1917, "is that it is our duty to make the far-reaching principles of the newer psychological methods as universally applicable as possible, and to accomplish this it is essential that they should be made usable under the conditions which actually confront us in dealing with our patients. This is worth striving for, even at the risk of being charged with superficiality."

Other papers of more than usual interest were the following: an early account of adiposis dolorosa, miliary aneurysm of the brain, diffuse degeneration of the spinal cord, senile trepidant abasia, injuries to the spinal cord, a new syndrome, progressive vagus-glossopharyngeal paralysis with ptosis, and a very early paper on neuroglia. In this he used the Mallory stain which, at that time, was just coming to be recognized to demonstrate fibrils. In addition, the passing of one of his colleagues always called forth a biographical notice, written in a charming style, not often equaled in medical literature.

Outside of his medical work Taylor had many interests. He wrote a number of papers on witchcraft, especially the medical aspect of the problem. In his later years he became a devotee of chess, and established an informal club in Boston which met frequently in his house. On the grounds of his summer home at Ipswich he built a small stone building, known as the "chess house", ingeniously devised, in the shape of a chess "castle" where he could play a quiet game surrounded by pine trees which he himself had planted many years before. He particularly enjoyed the quiet friendship of medical colleagues and others, and for many years he was a member of a small medical dinner club. He was a popular member of his class in Harvard College and often spoke at the reunion dinners. On October 14, 1897, he married Elsie Brewster Howe, in New York. She and two daughters survive him.

Such, in brief outline is the life of our friend. It tells some of the facts of his distinguished career, but it cannot more than hint at the character of a loyal friend, a charming gentleman, an accomplished scholar. None that knew him, from his colleagues in the medical school to the humblest servant, could or did fail to love him, and the better they knew him, the stronger became their affection and esteem. His love of his profession was great, but just as great was his interest in his other studies, such as witchcraft (in which subject his published studies gained him a membership in the American Academy of Arts and Sciences). His unfailing sense of humor, and his interest in the characters and the affairs, large or small, of his friends was delightful. He was honest and fearless. In his relations with others he personified kindness and consideration. To have known him intimately was a prize which his friends would exchange for few other privileges and satisfactions of life.

H. R. V.,
F. B. L.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

WATKINS, ROYAL P. A.B., M.D. Columbia University College of Physicians and Surgeons (Medical Department of Columbia University) 1892. F.A.C.S. Consulting Surgeon, Worcester City Hospital. Surgeon-in-Chief, Fairlawn Hospital, Worcester. His subject is "Chronic Appendicitis." Page 335. Address: 332 Main Street, Worcester, Mass.

STEELE, GEORGE L. M.D. University of Vermont 1914. F.A.C.P. Assistant Visiting Physi-

cian, Metabolist and Electrocardiographist, Springfield Hospital. Cardiologist, Wesson Memorial Hospital, Springfield, Mass. Consulting Physician, Shriner's Hospital for Crippled Children, Springfield, Mass., and Nobel Hospital, Westfield, Mass. His subject is "Medical Aspects of Appendicitis." Page 341. Address: 20 Maple Street, Springfield, Mass.

CLUTE, HOWARD M. B.S., M.D. Dartmouth Medical School 1914. F.A.C.S. Surgeon, Lahey Clinic. Assistant Surgeon, New England Baptist Hospital and New England Deaconess Hospital. His subject is "Factors Reducing Mortality in Acute Appendicitis." Page 347. Address: 605 Commonwealth Avenue, Boston.

MULLER, GEORGE P. M.D. University of Pennsylvania 1899. F.A.C.S. Professor of Clinical Surgery, University of Pennsylvania. Surgeon to University and Misericordia Hospitals. His subject is "Mortality of Acute Appendicitis." Page 355. Address: 1930 Spruce Street, Philadelphia, Pennsylvania.

ROSENFIELD, HAROLD H. M.D. University and Bellevue Medical School 1920. Gynecologist to Out-Patient Department, Beth Israel Hospital. Instructor in Gynecology, Tufts Medical School. Assistant Surgeon to the Gynecological Department, Boston Dispensary. Address: 483 Beacon Street, Boston. Associated with him is

DAVIDOFF, REUBEN B. B.S., M.D. College of Physicians and Surgeons, Columbia University 1922. Out-Patient Surgeon, Beth Israel Hospital. Teaching Assistant in Surgery, Tufts Medical School. Address: 483 Beacon Street, Boston. Their subject is "A New Procedure for Obstetrical Analgesia." Page 366.

MARVIN, FRANK W. A.B., M.D. Harvard University Medical School 1914. Assistant Visiting Anesthetist, Boston City Hospital. Visiting Anesthetist to Newton Hospital and Boston Psychopathic Hospital. His subject is "Pantocain in Spinal Anesthesia." Page 368. Address: 520 Commonwealth Avenue, Boston.

MISCELLANY

TUBERCULOSIS ABSTRACTS

BY THE NATIONAL TUBERCULOSIS ASSOCIATION

A NEW TUBERCULIN

MA100 is obtained from different types of tubercle bacilli and the timothy-grass bacilli. The protein is identical with the original tuberculo-protein isolated by Seibert. The bacilli are grown on Long's medium, a synthetic medium of fixed-standard composition, which is protein-free before culture. After growth the bacilli are removed by filtration and the protein of the medium is precipitated at a given isoelectric point by glacial acetic acid. Thus the identical substances are constantly brought down and these are not subjected to any denaturing process with heat or chemicals. The diluent was normal saline solution. Thus the testing substances contain no phenol or glycerine.

Four types of acid-fast bacilli were used, namely, the human, the bovine, and avian types of tubercle bacillus and the timothy-grass bacillus. The corresponding proteins obtained have been designated as *MA100 Human*, *MA100 Bovine*, *MA100 Avian*, and *MA100 Timothy*. The origin and virulence of each of the strains of bacilli from which the protein substances were derived, are known. The O. T. used for comparison also is of known derivation and sensitivity. Therefore, the reactions obtained can be safely compared and checked.

Old Tuberculin, as it is now marketed, is standardized on the basis of the minimum dose required to kill a tuberculous guinea-pig. It has recently been shown, however, that O. T. contains a polysaccharide which is equally as lethal for tuberculous guinea-pigs as is the acid-fast bacillary protein, but it produces no reaction when injected into the skin of tuberculous animals. Thus, standardizing tuberculin on the basis of the minimal lethal dose tells us nothing about its skin-testing potency. As a consequence, O. T. is known to differ as much as 400 per cent in that respect.

PROCEDURE

For this study, the dosage of O. T. was established as 0.01 mgm. The MA100 human protein was diluted with normal saline solution to give the same skin reaction as that obtained with the established dose of O. T. This proved to be 0.0005 mgm. If the skin reactions to these doses were negative at the end of 48 hours, the test was repeated with double the dose. The amount of MA100 of the several types necessary to produce a reaction comparable with that of the established O. T. was found to vary so that the following dosages were finally determined:

Tuberculin O. T.	0.01	mgm.
MA100 Human	0.0005	mgm.
MA100 Bovine	0.0001	mgm.
MA100 Avian	0.001	mgm.
MA100 Timothy	0.01	mgm.

QUESTIONS INVESTIGATED

Answers to five questions were sought.

1: *Is the MA100 human protein more sensitive and more selective than O. T.?*

A total of 2,328 individuals were tested simultaneously with O. T. and MA100 human. The group included general hospital patients, both adults and children, sanatorium employees ranging in age from 17 to 73, a general population group composed of nurses in general hospitals, medical students and school children in a rural section. The results are presented in detailed tables in the original article. It was found that more individuals reacted to the MA100 human protein than to O. T. and this was true of known cases of tuberculosis as well as in the general population group.

2: *Are the doses recommended safe or are dangerous reactions encountered?*

The reactions to the initial dose of O. T. and to the different MA100 proteins were compared. Reactions were recorded as plus one to four. Necrosis is associated only with a four-plus reaction and of these there were none in the series. There were no constitutional reactions in any of the tests but there were seven reactions which might be called disagreeable in that they were accompanied by lymphangitis or enlarged, painful axillary lymph nodes or superficial vesicles, which were not followed by or associated with necrosis. None of the reactions might be called dangerous. Two of the seven disagreeable reactions occurred with O. T. One with MA100 human protein, one with MA100 bovine protein, two with MA100 avian and one with MA100 timothy.

3: *Are the doses recommended and used in this study large enough or should they be increased?*

Of 540 clinically tuberculous individuals 91.1 per cent reacted to the established doses of MA100 human protein. In another group of 314 clinically tuberculous individuals 92 per cent reacted to both the initial and repeat doses of MA100 human protein. The 8 per cent of this group who failed to react either died or left the sanatorium before the test could be repeated.

By means of another series of tests, an attempt was made to determine whether or not a larger dose would yield a higher percentage of positive reactors. The authors conclude that the doses of the MA100 human protein as recommended (0.0005 mgm. and 0.001 mgm.) are apparently large enough and, therefore, need not be increased.

4: *Are the various MA100 proteins specific in that they will pick up only infections by the bacillus from which they are isolated?*

An analysis of the percentages of positive reactions in several thousand individuals tested with O. T. and the different MA100 proteins justified the authors in concluding that the bovine, avian and timothy MA100 proteins are not specific. They question whether O. T. in high concentration of a mgm. or more is specific. In small doses of O. T. and MA100 human protein, the reaction is apparently specific.

5: *Is there a protein substance common to all acid-fast bacilli?*

Comparison of reactions from O. T. and the several MA100 proteins lead the authors to conclude that there is apparently a protein substance common to all acid-fast bacilli, which if given in large enough doses will call forth a typical tuberculin reaction in just as large a proportion of the people as does O. T.

CONCLUSIONS

1. The MA100 human protein is as sensitive and as selective as O. T., and probably more so.

2. The initial and repeat doses recommended for the MA100 human protein are safe in that dangerous reactions are not encountered.

3. The initial and repeat doses recommended for the MA100 human protein are large enough to pick out the majority of tuberculous individuals and apparently do not need to be increased.

4. The MA100 proteins are apparently not specific in large doses at least.

5. There is apparently a protein substance common to all acid-fast bacilli, which, if given in large enough doses, will elicit the same type of reaction as that obtained from Old Tuberculin.

6. As the MA100 protein represents a substance in a purified form which can always be reproduced at the same isoelectric point and which can be weighed out in milligram doses, so that the exact milligram content of the solution is known, it is a better testing substance than Old Tuberculin.

Among the workers mentioned by the authors as participators in the study are Elmer H. Funk, F. M. Huntoon, H. J. White, J. Aronson, Fred Carter, Everett K. Geer, Harold Diehl, D. R. Hastings, Florence Seibert, Parke, Davis & Co., H. K. Mulford Laboratories, Peter Masucci, Kenneth L. McAlpine, Esmond R. Long, Professor H. Valée, L. Van Es, Edward R. Baldwin, M. Dorset, M. I. Smith, W. H. Morriss, John Reichel, National Institute of Health.

The Present Status of the Skin Reaction in Tuberculous and Non-tuberculous Subjects, E. S. Mariette and E. P. K. Fenger, in collaboration with Elmer H. Funk, F. M. Huntoon and H. J. White. Am. Rev. of Tuberc., March, 1932.

A RADIO MESSAGE PREPARED AND SPONSORED BY THE COMMITTEE ON PUBLIC EDUCATION OF THE MASSACHUSETTS MEDICAL SOCIETY FOR THE DEPARTMENT OF PUBLIC HEALTH

HEADACHE*

BY WILLIAM G. LENNOX, M.D.

Headache is no modern symptom, but apparently is as old as mankind. Skulls of prehistoric man have been found in which openings had been chiseled some months or years before death. It is not improbable that in some instances these openings were designed to relieve pains in the head, possibly with the view of releasing imprisoned evil spirits. Anyone who has experienced "splitting" headaches would willingly subscribe to the idea that devils are at work within.

Headache is one of the most common and troublesome of symptoms. Probably it is not now so frequent as some decades ago. Present improved methods of ventilation and heating have reduced noxious gases; improved lighting and more general correction of errors of vision have reduced eyestrain; and emphasis on fresh air and exercise have improved physique. Against these favorable conditions must be arrayed the present increasing hurry and strain of living which tend to increase all sorts of nervous symptoms.

Headache is not a disease but is a symptom or manifestation of various different disorders. Some

*Broadcast: April 5, 1932. Time: 4:20 P.M. Place: Hotel Bradford.

of these are trivial; some serious. In any case, a headache should be looked on as a red lantern, something which requires caution and investigation. There is, of course, an underlying pathology which is the immediate cause of all headaches. Brain tissue itself is insensitive, but tension of overlying membranes or stimulation of the nerves lying on the arteries of the brain may account for the pain.

Of the various conditions which may be associated with headache, those which involve injury or disease processes within the skull are the most serious. Such headaches are likewise most severe and intractable. Of first importance are infections which involve the membranes covering the brain, viz., meningitis or so-called "brain fever". There are several forms of meningitis, depending on the germ which causes the inflammation. Epidemic meningitis, tuberculosis, and syphilis are the most frequent. Infections in the brain itself, such as abscess or sleeping sickness, also cause headache. Tumor of the brain produces a very severe headache, with vomiting and loss of vision. Injuries and concussions of the brain unhappily are on the increase because of automobile accidents. The headaches which result sometimes persist for months after the person has in other respects recovered.

Abnormalities in the skull itself are important sources of chronic headache. There are sinuses or cavities above and below the eyes and behind the nose. These have tiny passages which lead into the nasal cavity. The passages and subsequently the sinuses may become infected as a result of persistent head colds. If the passages into the nose are blocked, pus accumulates and the resulting pain is most uncomfortable. Headache which comes on after use of the eyes is very probably due to strain of the muscles which keep the eyes in focus. An infected root of a tooth or an unruptured tooth may also cause localized pain. Neuralgia of certain nerves of the face may cause pains resembling headache.

One must not think that a headache is indicative of disorders which lie always and only in the head. The burglar alarm does not necessarily sound at the point where the thief is entering the building; just so, the headache may signal danger to some portion of the body distant from the head itself.

A person with kidney or Bright's disease has headaches which may be present on waking in the morning. Headache in a pregnant woman may warn of impending eclampsia. In elderly persons hardening of the arteries may be associated with pains in the head. More constant and distressing, however, are the headaches which often attend high blood pressure. If the blood is either too poor or too rich in red cells, headache may be one of the accompanying symptoms. In addition many of the severe infectious diseases; typhoid fever, pneumonia, influenza, scarlet fever, have headache as a prominent symptom.

In all these conditions headaches are persistent and tend to continue or to recur at short intervals until the underlying disorder is corrected. In the great majority of persons, however, headaches are temporary or recur only at rare intervals or when there is a break in the routine of healthy living. Thus, many individuals have headaches if they become constipated, if they have been in an inadequately ventilated room, or have been worried, hurried or emotionally upset. A headache may be the end product of fatigue of muscles and of mind.

Poisons must not be forgotten. The finding of the guilty agent may require "Sherlock-Holmes-like" activity. One of the most serious of headache producing poisons is carbon monoxide, present in illuminating gas, in the exhaust gas of automobiles, in furnace gas and even in tobacco smoke. An insidious feature is the fact that in the case of the automobile the exhaust gas in weak concentrations is odorless. Long-continued breathing of air containing a small amount of gas is as harmful as brief breathing of a more concentrated mixture. If the amount of carbon monoxide is small, persistent dull headaches may be the only evidence of trouble. The "hang-over" headache which follows overindulgence in alcoholic beverages is doubtless due to swelling of the brain as a result of saturation of brain cells with alcohol. Tobacco smoke in certain individuals produces headache. Likewise various drugs, such as nitroglycerine and certain coal tar products, may cause headache in susceptible persons.

We have not yet exhausted the conditions accompanied by headache. In many instances the cause is not physical but emotional. This is the "headache of convenience". There may be an unpleasant task to perform. An incapacitating headache is an effective means of escape. Such an ache may be entirely fictitious, the "pure" white lie of society, but also it may be an unconscious reaction to a trying situation. A headache may be the visible result of invisible conflicts of emotions or of desires. These so-called "nervous headaches" in neurotic individuals or in those poorly adjusted to their mental or social environment are real in the distress which they produce and real in the work which is required for their unraveling. Striking examples of persistent headache as a result of emotional trauma were found among the shell-shocked soldiers in the war. The performance of duties against which the inner man revolted caused such inner turmoil that various incapacitating symptoms resulted. In civil life there are numerous instances of what might be called domestic "shell shock", persons who are unhappily mated, who face apparently insoluble problems or who use physical disability as a means of holding the center of the stage.

One type of headache has such distinctive features as to give it a name. This is migraine or so-called "sick headache". Migraine is distinctive in that it often exists in families. It comes at periodic intervals, is so severe as to interfere with work, and is associated with nausea or vomiting.

Oftentimes, visual or other disturbances give a warning of the approach of an attack. Sometimes one side of the head only is involved. Attacks of migraine and of epilepsy have features of resemblance. Both are particularly difficult to treat; both call for further research investigation. A clinic for the study of persons who suffer with migraine has been established recently in the Outpatient Department of the Boston City Hospital.

As for the treatment of headache, it is clear that the essential thing is the determination of the cause. For those occasional dull headaches which are the obvious results of errors of living, a period of rest and quiet, with the eyes covered and cold applied to the head, will be sufficient.

For frequently recurring or persistent headaches the case is different. The cause must be searched for. The person with a headache is inclined to run to the medicine closet. If the headache recurs frequently, such action is similar to covering the red lantern which warns of danger. From the character of the headache itself, it is difficult to say whether the cause is a serious or a trivial one. Furthermore, the cause of a headache may differ at different times, and also it may be the result of a combination of untoward conditions. Thus, a person with high blood pressure may also have eyestrain. In this case the fitting of glasses will only partially relieve the situation. For these reasons the sufferer from headaches should first consult a physician and have a thorough going over including examination of the urine and measurement of the blood pressure. Only in this way is it possible to discover whether life as well as comfort is menaced.

Treatment of headache should be directed at removing the cause. This may involve anything from an operation for the removal of a brain tumor, to relief of constipation. Drugs are to be used only as a palliative measure for temporary relief. In recent years there has been widespread advertisement and sale of drugs for the relief of headache. Dependence on these remedies is to be deprecated. Though not habit-forming in the sense that drugs like morphine and cocaine are, the habit of using sedative drugs may prove harmful because over-dosage may result. In any case, habitual use of headache powders or pills is a poor way of treating a valuable danger signal. Naturalists tell us that the ostrich does not, as he has been reported to do, bury his head in the sand at the approach of danger. There are, however, many human beings who do this, when they habitually treat a headache by means of a sedative drug.

CORRESPONDENCE

A QUESTION OF PROCEDURE

August 16, 1932.

Editor,
The *New England Journal of Medicine*,

"A Prophet is without Honor in his own Country." Page 579 of the August 13, 1932, issue of the *Journal of the A. M. A.* indicates that Honor? is due the Massachusetts Medical Society.

Why must a group of honest and conscientious physicians have such a black sheep as David L. Martin among them?

Either his or my name goes off the Massachusetts Medical Society's roster.

Yours truly,
NOEL G. MONROE.

39 Boylston Street, Boston, Mass.

A CORRECTION OF THE STATEMENT RELATING TO MISS HARRIET DELAMERE

Collis P. Huntington Memorial Hospital
Harvard Medical School
Boston

August 12, 1932.

Editor, *New England Journal of Medicine*,

I should like to make a correction in regard to the tribute to Miss Harriet Delamere, which appeared in your last edition. Miss Delamere is a graduate of the Boston City Hospital, Class 1910.

Very truly yours,
ANNA L. GIBSON, R.N., *Superintendent*.

DISSATISFACTION WITH CIVIL SERVICE EXAMINATIONS

The *New England Journal of Medicine*,
8 The Fenway, Boston.

The tribute, as expressed in your *Journal* as of August 11, to the thirty-six physicians who took the civil service examination for medical inspector of the Boston Health Department is no doubt appreciated by all. You were good enough to mention the name of one and give his qualifications. Why didn't you give the names of all and state their qualifications? What you do for one should be done for all.

At this time I desire to enlighten the members of the medical fraternity that civil service examinations, when it comes to medical positions, are practically a farce. One may pass with 100% on his written, but when it comes to his experience one's average can very easily be lowered and last but not least when it comes to the oral, so to speak, because at the oral you are asked your name, address, age, experience and whether you are a veteran. I have been informed that no one ever received more than 90% and many received from 70% upward. This oral is the biggest farce. I take it for granted that the commissioners must have some definite points upon which to give one a rating on his oral interview. If medical questions were asked, I could account for the mark but when nothing is asked, I fail to see how one can come to a conclusion of 70% or over.

In order to be fair to all applicants, the oral should have no standing except when specific questions pertaining to the position are asked.

It is about time that the medical profession should take more interest and help to frame and pass

proper rules for medical examinations as given by the civil service and, by so doing, politics and other influences can be done away with. Each and every applicant should have the same opportunity.

I am glad to state that I was one of the thirty-five physicians who passed the civil service examination for medical inspector and received only 86% in my written, although after reëxamining my papers, I could very easily have had it raised a few points. However, it would have made very little difference in my final standing.

Very truly yours,

BERNARD ZUCKERMAN, M.D.

RECENT DEATH

TAYLOR—DR. EDWARD WYLLYS TAYLOR died at his home, 457 Marlborough Street, Boston, August 17, 1932, after a long illness.

Dr. Taylor was born in Montclair, New Jersey, and received the degrees of A.B. and A.M. from Harvard University and the M.D. degree from the Harvard Medical School in 1891. His early activities were centered in teaching neuropathology at the University of Vermont and the Harvard Medical School, holding the position of Professor of Neurology at Harvard from 1917 until his death, and the James Jackson Putnam Professorship from 1920 until 1926 when he resigned to become Professor Emeritus. He was Assistant Editor of the *Boston Medical and Surgical Journal* from 1899 to 1912 and Editor-in-Chief from 1912 to 1915. He also held the position of Associate Editor of the *Journal of Nervous and Mental Diseases* for several years.

In preparation for his specialty, Dr. Taylor studied in Berlin and Freiburg, Germany.

Dr. Taylor joined the Massachusetts Medical Society in 1894 and was a member of the Standing Committee on Publications for several years, serving in that capacity at the time of his death.

He was a Fellow of the American Medical Association, the American Association for the Advancement of Science of the Massachusetts Medical Society, and a member of the American Neurological Association, the American Association of Pathologists and Bacteriologists, the Boston Society for Medical Improvement and various other bodies. While at Harvard he belonged to the Signet Society and the Hasty Pudding Club. He was a member of the University Club of Boston.

Dr. Taylor is survived by a widow, the former Elsie Brewster Howe of New York; two daughters, Mrs. Powell Robinson of Montclair, N. J., and Miss K. B. Taylor of Boston; and a brother, Alfred Taylor of Wakefield, Rhode Island.

NOTICE OF MEETING

AMERICAN ACADEMY OF PHYSICAL THERAPY

The American Academy of Physical Therapy will hold its annual meeting at the Hotel Walton in Philadelphia on October 12, 13 and 14, 1932. Besides the program of scientific papers, clinics will be held at the various hospitals. There will be a manufacturers' exhibit.

ARTHUR H. RING, *Secretary.*

SOCIETY MEETINGS
CONGRESSES AND CONFERENCES

September 19-22 — International Congress of Biliary Lithiasis. See notice on page 427, issue of February 25.

September 26, 27, 28—Eighth Clinical Congress of the Connecticut State Medical Society. See page 331, issue of August 18.

September 30, October 1—Scientific Program for Annual Meeting of the New England Surgical Society. See page 333, issue of August 18.

October 12, 13 and 14—American Academy of Physical Therapy. See notice elsewhere on this page.

October 17-21—Clinical Congress of the American College of Surgeons. See page 1330, issue of June 23.

October 17-28—The 1932 Graduate Fortnight of the New York Academy of Medicine. See page 1375, issue of June 30.

October 22-24—Health Education Institute. See not on page 47, issue of July 7.

October 24-27 — American Public Health Associati See notice on page 1321, issue of December 31, 1931.

October 24-28—Inter-state Postgraduate Medical Association of North America. See page 39, issue of January 7.

BOOK REVIEWS

Early Science in Oxford. By R. T. GUNTHER. Vol. IX, De Corde by Richard Lower, London, 1669, with introduction and translation by K. J. Franklin. Oxford, 1932.

This is the ninth volume of a splendid series of reprints published by R. T. Gunther from the Old Ashmolean, Oxford, on early science. These volumes have contributed greatly to our knowledge of chemistry, mathematics, physics and medicine as understood by the important scientific men who lived in Oxford in the seventeenth and eighteenth centuries. This volume is a reprint of a treatise on the heart by Richard Lower, M.D., printed in facsimile from the 1669 edition, with an excellent translation by Dr. K. J. Franklin; a brief biographical notice is added. There is a definite need for this translation of a book second in importance only to that of William Harvey. The work was done at the instigation of Professor John F. Fulton, now of Yale University. The facsimile is beautifully reproduced and the translation excellent. This book, as may be said of the others in the series, is indispensable to anyone working in the field of medical history.

Some details about Lower and his book are to be found in a short paper by Dr. Franklin in the *Annals of Medical History* for December, 1931.

A Text-Book of Medicine for Nurses. By E. NOBLE CHAMBERLAIN. Published by Humphrey Milford, Oxford University Press.

This is a textbook of medicine for nurses containing a very practical and comprehensive presentation of the subject for the student nurse of today. With the advancement of education for nurses Dr. Chamberlain realizes that a cursory statement of the causes and effects of disease is not sufficient.

The chapter on "Diseases of the Stomach and Intestines", and the one on "Diseases of the Urinary Organs", are particularly well done and the cuts are excellent.